pp. 947–1052] [December, 1945

TROPICAL DISEASES BULLETIN

Vol. 42.] 1945. [No. 12.

MAIN CONTENTS :

	PAGES
Urinary Amoebiasis : A Critical Review	947–959
Malaria	959–968
Blackwater Fever	968
Trypanosomiasis	969–976
Leishmaniasis	976–979
Fevers of the Typhus Group	979–996
Bartonellosis	996
Dengue	996
Bacillary Dysentery ...	997–998
Amoebiasis and Intestinal Protozoal Infections ...	999–1002
Relapsing Fever and other Spirochaetoses ...	...1002–1003
Yaws	...1003–1004
Leprosy	...1004–1015
Helminthiasis ...	...1015–1021
Sprue...	...1021–1022
Haematology ...	...1022–1025
Venoms and Antivenenes ...	1026
Dermatology and Fungous Diseases	...1026–1030
Tropical Ophthalmology	...1030–1037
Tropical Ulcer ...	...1037–1038
Miscellaneous Diseases	...1038–1042
General Entomology	...1042–1046
Laboratory and Technical Procedures ...	...1046–1048
Reports, Surveys and Miscellaneous Papers ...	...1048–1049
Book Reviews ...	...1049–1052

For detailed Contents see pp. ii, iv, v, vi and viii.

Issued under
the Direction of the
Honorary Managing Committee of the
BUREAU OF HYGIENE AND TROPICAL DISEASES,
Keppel Street, London, W.C.1.
(Telephone: MUSEUM 3041)

Price 2/6 Net.] *Entered as Second Class Matter in the U.S.A. Post.* Annual Subscription 21/-

HONORARY MANAGING COMMITTEE

APPOINTED BY THE SECRETARY OF STATE FOR THE COLONIES.

Chairman: W. H. Kauntze, *C.M.G.*, *M.B.E.*, M.D., D.P.H., F.R.C.P. (*Chief Medical Adviser to the Secretary of State for the Colonies*).

Wm. Horner Andrews, D.Sc., M.R.C.V.S.

Lieut.-General Sir Ernest W. C. Bradfield, *K.C.I.E.*, *O.B.E.*, M.S., F.R.C.S.Ed., I.M.S. (retd.).

Professor Patrick A. Buxton, M.A., M.R.C.S., L.R.C.P., D.T.M. & H., F.R.S. (*representing the Royal Society*).

Sir Wilson Jameson, *K.C.B.*, LL.D., M.D., F.R.C.P. (*representing the Ministry of Health*).

Professor J. M. Mackintosh, M.D., F.R.C.P., D.P.H.

Sir Philip Manson-Bahr, *C.M.G.*, *D.S.O.*, M.D., F.R.C.P., D.T.M. & H.

Sir Edward Mellanby, *K.C.B.*, M.D., F.R.C.P., F.R.S., K.H.P. (*representing the Medical Research Council*).

E. R. A. Merewether, M.D., M.R.C.P., F.R.S.E., K.H.P.

J. B. Sidebotham, M.A. (*of the Colonial Office*).

Secretary: C. A. Grossmith, *O.B.E.* (*of the Colonial Office*).

STAFF OF THE BUREAU:

Acting Director: Charles Wilcocks, M.D., M.R.C.P., D.T.M. & H.

Acting Assistant Director: J. F. Corson, *O.B.E.*, M.D., D.P.H., D.T.M. & H.

Secretary: R. L. Sheppard.

LIST OF ABSTRACTERS IN THIS ISSUE.

A. R. D. Adams, M.D., Ch.B., M.R.C.P., D.T.M.

J. J. C. Buckley, D.Sc.

J. R. Busvine, Ph.D., B.Sc.

Professor P. A. Buxton, M.A., M.R.C.S., L.R.C.P., D.T.M. & H., F.R.S. (*Sectional Editor*).

J. F. Corson, *O.B.E.*, M.D., D.P.H., D.T.M. & H.

J. C. Cruickshank, M.B., Ch.B., D.T.M.

W. A. L. David, B.A.

L. J. Davis, M.D., M.R.C.P.

J. D. Fulton, M.A., B.Sc., Ph.D., M.B., Ch.B., D.T.M.

F. Hawking, D.M., D.T.M.

Professor Edward Hindle, M.A., Sc.D., Ph.D., F.R.S. (*Sectional Editor*).

Lieut.-Col. E. W. O'G. Kirwan, *C.I.E.*, M.Sc., M.D., F.R.C.S.I., I.M.S. (retd.).

H. S. Leeson, F.R.E.S.

Brigadier R. M. B. MacKenna, M.D., F.R.C.P., R.A.M.C.

Sir Philip Manson-Bahr, *C.M.G.*, *D.S.O.*, M.D., F.R.C.P., D.T.M. & H.

Maj.-Gen. Sir John W. D. Megaw, *K.C.I.E.*, D.Sc., M.B., B.Ch., I.M.S. (retd.) (*Sectional Editor*).

Lt.-Col. F. Murgatroyd, M.D., F.R.C.P., R.A.M.C.

Maj.-Gen. Sir Leonard Rogers, *K.C.S.I.*, *C.I.E.*, M.D., F.R.S., I.M.S. (retd.). (*Sectional Editor*).

Sir H. Harold Scott, *K.C.M.G.*, M.D., F.R.C.P., D.P.H., D.T.M. & H.

H. W. Swann, M.B., B.S.

C. M. Wenyon, *C.M.G.*, *C.B.E.*, M.B., B.S., B.Sc., F.R.S. (*Sectional Editor*).

F. Norman White, *C.I.E.*, M.D., D.P.H., I.M.S. (retd.) (*Sectional Editor*).

Charles Wilcocks, M.D., M.R.C.P., D.T.M. & H.

BUREAU OF HYGIENE AND TROPICAL DISEASES.

TROPICAL DISEASES BULLETIN.

Vol. 42.] 1945. [No. 12.

URINARY AMOEBIASIS.

(A Critical Review.)

By J. M. Watson, A.R.C.S.

Wellcome Laboratories of Tropical Medicine.

Historical Survey.

Urinary amoebiasis is rather a loose definition of amoebic infection in various parts of the urinary tract. The question of amoebic infection of the urinary tract has for long been vexed. At one time it was considered to be a relatively common condition, especially in Egypt and South America. Castellani and Chalmers (1919) regarded amoebic pyelitis, cystitis and urethritis as definite pathological and clinical conditions. Certain continental workers, especially, published a considerable number of records of alleged cases, most of which, however, are now considered to be of dubious authenticity. Such authorities as Craig and Faust (1943), Stitt (1942) and Manson-Bahr (1940), in discussing amoebiasis, dismiss amoebic invasion of the urinary tract as one of the rarest complications of the disease.

The earliest record of amoebae in the urine was made by Baelz (1883), who found actively motile forms in enormous numbers in the urine of a young Japanese woman suffering from tuberculosis of the lungs and urogenital apparatus, who died shortly after entering hospital. The urine, which was removed by catheter, contained pus, blood and necrotic tissue. The amoebae, which were about 50μ in diameter, had granular cytoplasm and a vesicular nucleus and are stated to have been in every way similar to Lösch's "Amoeba coli." Baelz suggested that they represented a new species, for which he tentatively proposed the name "Amoeba urogenitalis," but there seems little reason to doubt that they were actually *Entamoeba histolytica.* Unfortunately, no examination of the faeces was made, so that the existence of a concomitant intestinal infection remains in doubt.

Subsequently Jurgens (1892), Kartulis (1893), Posner (1893), Wijnhoff (1895) and McDill and Musgrave (1905) claimed to have found amoebae in human urine, but it seems probable, from the somewhat inadequate accounts which they give, that the bodies they observed were not Protozoa.

Craig, in 1911, recorded an undoubtedly genuine case of urinary amoebiasis, but in this instance there was a heavy intestinal infection and the amoebae were frequently discharged into the urine through a fistula which had formed

between an ulceration of the rectal wall and the wall of the bladder to which it was adherent. There was therefore no actual infection of the urinary tract in this case.

Cases of amoebic infection of the urinary tract have also been reported by JEFFRIES in 1904 (according to FANTHAM, 1916) and by SIMONIN in 1914 (according to FIESSINGER and PARTURIER, 1926), but the reviewer has not been able to trace these papers.

A somewhat dubious case was recorded from Shanghai by FISCHER (1914), which, from the description, may possibly have been authentic, but unfortunately insufficient details are given to enable a decision to be made. Also in 1914 LYNN reported amoebae, blood and pus in the urine of a patient who is stated to have used the same syringe for washing out both the rectum and the bladder ; but as no amoebae were found in the stools, this case is extremely doubtful.

In 1915, WALTON published an account of what seems to be one of the few indubitably genuine cases of urinary amoebiasis. Numerous actively motile amoebae were found in the urine of a male Indian suffering from haematuria and anasarca of the legs, penis and scrotum. The urine was scanty, blood-stained and of " acid reaction," with a specific gravity of 1013. The patient, who was considered to be a case of chronic Bright's disease, gave no history of previous dysentery, but amoebae were found in the stools. A careful description is given of the amoebae from the urine, from which it seems clear that the organisms in this case really were living specimens of *Entamoeba histolytica*, although from the dimensions given they must have represented an unusually large strain. Injection of the urine into the rectum of kittens was, however, without result. Walton remarks that " probably an abnormal condition of the urine is necessary for the parasites to establish themselves."

MACFIE (1916) reported five cases of urinary amoebiasis from the Gold Coast, of which one only is adequately described ; from his account it seems possible that the organisms which he found in this instance were *Entamoeba histolytica*. Dubious cases were reported about this time by WARD, COLES and FRIEL (1916), FRIEL (1917), WRIGHT (1917), and CHALMERS and O'FARRELL (1917). According to FIESSINGER and PARTURIER (1926), KOSTER (1918) collected fourteen cases of urinary amoebiasis, while BARTLETT (1917) found the condition to be common, but the reviewer has not been able to consult the original paper. In all these instances the evidence for the occurrence and identity of the parasite in the urine is inadequate for a decision to be reached. It seems likely, as DOBELL (1919) has remarked, that diagnosis rested on errors of observation and interpretation.

In 1918 ARAVANTINOS and MICHAILIDIS recorded the occurrence of active, uninucleate amoebae in the urine of a Greek boy suffering from cystitis. No figures or description of the alleged amoebae are given, but the authors did not consider that they were *E. histolytica*, and DOBELL (1919) has suggested that they were probably degenerating forms of *Trichomonas vaginalis*.

DOBELL (1919), in discussing the question of urinary amoebiasis, came to the conclusion that there was no reason why *Entamoeba histolytica* should not occasionally occur in the urine, but that such infection of the urinary tract must always be secondary to a primary infection of the large intestine. This author considered that of the cases recorded up to that date the only one which could be regarded as undoubtedly genuine was that of Walton.

Since the publication of Dobell's monograph a large number of cases, most of them of very doubtful authenticity, have been recorded. BAYMA and FAGUNDES (1918) published a brief note of a case of acute cystitis in which numerous active amoebae and cysts occurred both in the urine and in the stools but no details are given. NOGUE (1919) found what he believed to be

amoebae in the urine of a schistosomiasis patient who was suffering concurrently from amoebic dysentery. LEGER is stated to have identified these organisms as *E. histolytica*, and the genuineness of this case is therefore hardly in doubt. There is a reasonable possibility that amoebae from rectal or intestinal lesions may have reached and passed through the bladder wall in the wake of the schistosome eggs. DE MELLO (1919) recorded a doubtful case. JACKSON in 1922 described an amoeba-like organism, found at autopsy in the uriniferous tubules of the kidneys from a 15-months child who had died, in convulsions, from broncho-pneumonia and parenchymatous nephritis. Active division of these organisms was apparently going on in the tissues, but neither the figures nor the description suggest that they were actually amoebae.

In the next few years a spate of papers describing alleged cases of urinary amoebiasis issued from Alexandria. Foremost protagonists of the alleged disease were PETZETAKIS (1923 a & b ; 1924 ; 1925 a, b & c), PANAYOTATOU (1924 ; 1926 a & b ; 1930) and RALLI and PANAYOTATOU (1923). No useful purpose would be served by considering each of these papers separately, especially as some of them are repetitive. These authors describe cases of cystitis and nephritis which are stated to be of amoebic origin, although in only a few of them was there any history of previous or concurrent amoebic dysentery. Living and dead entamoebae—in some cases showing active protrusion of pseudopodia, and containing red blood corpuscles—and occasionally also cysts, are stated to have occurred in the urine. In all these cases, apparently, a cure was effected by intravenous injection of emetine, and this was considered to be a further reason for identifying the organisms in question as *E. histolytica*. Petzetakis, in particular, was fully aware of the possibility of mistaking large phagocytic cells for amoebae, and in one of his publications (1925 b), in which he discusses at length the whole situation with regard to this infection, he refers to this point. On the other hand, although in two cases described in the same publication he made permanent preparations of the supposed amoebae, using Heidenhain's iron haematoxylin, the results were evidently poor since he gives no figure of the stained specimens and states that he considers this method of identification as unsatisfactory, identification of the amoebae in the living condition being preferable. WENYON (1926) considered these cases to be genuine, but this seems doubtful in view of the fact that intrarectal injection of the urinary sediment (not urine, *cf.* WALTON) into a cat failed to produce any amoebic infection. The other cases recorded by Petzetakis are less fully documented, but in some of them (1923 b) acute amoebic dysentery also occurred, and in spite of the fact that no satisfactory figures are given, it must be considered that these may have been genuine, although the possibility of the urine having become contaminated from the faeces does not appear to have been excluded. In 1924, and again in 1925, Petzetakis described a new species, *Entamoeba polymorpha*, from the urine of two patients suffering from urethritis, but the inadequate description and crude figures make it seem very doubtful whether, in fact, these organisms were Protozoa. In one case described by PETZETAKIS and PETRIDIS (1925), amoebae were alleged to occur in the urine of a patient infected with *Schistosoma haematobium*, and, as usual, emetine treatment is claimed to have eliminated them from the bladder ; but, as pointed out above, in such a case the amoebae may have reached the bladder from rectal ulcerations through lesions caused by the schistosome eggs. PETZETAKIS and MYLONAS (1924) described the form of ulcerations in the bladder believed to have been produced by amoebae.

With regard to the cases reported by PANAYOTATOU (1924, 1925 a & b) it would seem that some of them at least may have been genuine, since centrifuged deposits from specimens of the urine containing the supposed amoebae

were injected intrarectally into kittens, producing a fatal dysentery with typical lesions. Unfortunately, however, from the account given, at any rate in the earlier paper, it is not certain that these experiments were carried out with due precautions and under fully controlled conditions, and they must therefore be regarded with a certain amount of doubt, especially in view of the comments later put forward by KHOURI (1929) and FARMAKIDIS (1930), working in the same locality (see below).

In considering the instances of amoebae occurring in the urinary tract recorded by Petzetakis and Panayotatou and their collaborators, it should be borne in mind that they regarded amoebiasis not as a localized infection of the intestine but as a generalized infection, spreading throughout the body by the blood and lymphatic streams, to which PETZETAKIS (1925) applied the name "amibhémie." Of this malady they considered amoebic dysentery to be the most frequent and best known of the various local manifestations which could arise. However, it is now a well-established fact that amoebiasis is primarily an infection of the bowel, from which it may spread in metastatic fashion, especially to the liver and sometimes to other organs.

Another protagonist of the occurrence of urinary amoebiasis was FRANCHINI (1925 a & b; 1926). In several if not all of the cases recorded by this worker there was a concurrent intestinal infection of actual amoebic dysentery and, as in all such cases, the possibility exists that the amoebae may have gained access to the urinary tract by a fistula between the bladder and the ulcerated rectum. The possibility of contamination of the urine was said to be definitely excluded.

According to PETZETAKIS (1925–6) and GIROLAMI (1936) other records of cases of urinary amoebiasis were published in 1924 by PEYROT, CHEYSSIAL and ARLO, but unfortunately it has not been possible to consult the original papers of these workers, and they must therefore be passed over without comment. REISS (1924) recorded two cases of what he believed to be amoebic infection of the bladder, and regarded the organisms in question, for what seem to be totally insufficient reasons, as a new species of *Entamoeba*. As in one of his cases there was a previous history of amoebic dysentery, and as cystoscopy revealed ulceration of the bladder wall, this instance may well be genuine. Somewhat dubious cases of amoebic infection of the urinary tract were also recorded in 1924 by CARBONARO, and by WORSLEY and BATEMAN, in both of which it was considered by the authors that the kidney was possibly infected, but the evidence is unsatisfactory. VICHREV (1925) recorded a case of infection of the vagina and bladder with "Amoeba urogenitalis," and other alleged cases of urinary amoebiasis were reported by BUCHMANN (1925) from Palestine, and by BALSAMELLI (1926) and VIVIANI (1926) from Italy.

EVANS (1926), in an important paper on the surgical aspects of amoebiasis, throws doubt on the allegedly frequent occurrence of infection of the urinary tract with *E. histolytica*, remarking that—apart from direct extension of the liver—amoebic abscess of the kidney has not been recorded. This author also states that he has never met with a genuine case of amoebiasis of the genito-urinary system and that he regards this part of the body as exceptionally resistant to amoebic infection.

Records of further cases of urinary amoebiasis continued to be published, particularly by workers in the Mediterranean region and in South America. CASTEX and GREENWAY (1926) reported the occurrence of amoebae in the urine from cases of nephritis. FIORITO (1926) recorded a possibly genuine case of amoebic cystitis in a female who had suffered from amoebic dysentery for three years, and in whom deep intestinal ulceration had favoured the passage of the amoebae to the bladder by the lymphatic route. FIESSINGER and PARTURIER (1926) published a further detailed account of a possibly

genuine case, together with a useful historical review. GOYENA (1926) also recorded what might have been an authentic case, in which vegetative and cystic forms of *E. histolytica* occurred in the urine of a dysentery patient; from this account there seems to be a distinct possibility of a fistula from the intestine to the bladder having permitted the passage of the amoebae, but, as usual, the evidence is insufficient. A somewhat similar case was recorded by PAWAN (1926).

An especially valuable contribution to the problem of urinary amoebiasis was made in 1926 by CARVAILLO and SAUTET. These workers were led by the increasing number of reported cases of urinary amoebiasis to attempt to reproduce the disease experimentally. Using kittens, which, as is well known, are peculiarly susceptible to infection with *E. histolytica*, they sealed the urinary meatus with a collodion plug, performed a median suprapubic cystotomy and inoculated cultures from a case of amoebic dysentery directly into the bladder. The collodion seal was removed after two hours. Although a number of such experiments were made, none of the experimental animals developed any symptoms of urinary amoebiasis. At subsequent autopsy no bladder lesions were found (except one ulcer, which did not contain amoebae), no amoebae were found in the urinary tract, nor did any amoebae occur in the urine between inoculation and autopsy. From these results it is clear that *E. histolytica* does not readily establish itself in the bladder of cats.

YOKOCHI (1926) stated that "amoeba" could not live in urine. Unfortunately, only an abstract of this paper was available, and as the species of amoeba and the limit of concentration of the urine are not stated, this evidence is very inconclusive. As the orginal paper appeared in a medical journal, however, it seems likely that the amoeba used was one of the human intestinal species.

SITSEN (1927) published an account of a case of amoebic abscess of the kidney, discovered at autopsy. He discusses the numerous reports of amoebic infection of the urinary tract, points out that many of these must be regarded as not satisfactorily proven on the evidence adduced, and concludes that the amoebae may transfer from one site to another in the body *via* the lymphatic vessels.

A number of dubious cases of urinary amoebiasis were recorded by CHEREFEDDIN (1927), ROSSI (1927), VAN CUTSEM-FRANCO (1928), ROCCA (1928), PIRAMI (1928), SAUJEON (1928) and CHEVALIER and SOULIÉ (1928), in several of which there was no history of previous or concurrent dysentery. The last authors were alive to the possibility of mistaking haematophagous leucocytes for amoebae, and realized that urinary amoebiasis was always a secondary condition, hence it is possible that some of their cases were genuine. However, the principal evidence for the identification of *E. histolytica* in all these cases seems to have been ostensible cure of the condition by emetine injections. GUIDICEANDREA (1928) mentions an alleged case of combined bronchial and urinary amoebiasis showing abundant active amoebae in both urine and sputum.

In 1928 DESCHIENS and MELNOTTE reviewed the whole situation relative to amoebic infection of the urinary tract and cast grave doubts on the numerous reported cases of amoebic nephritis, cystitis and urethritis. They pointed out that diagnosis on the grounds of either the occurrence of alleged amoebae in urine or the curative effect of emetine is completely inadequate; that the identification of *E. histolytica* is not easy even for specialists, various other cells, and in particular large haematophagous leucocytes, being readily confused with it unless differential staining with iron haematoxylin after wet fixation is carried out; and that emetine, when injected intravenously, is beneficial in a variety of conditions, especially haemorrhagic and congestive

conditions, exercises a mild antiseptic and bactericidal action, and cannot be considered as exclusively amoebicidal. In Fez, a notorious centre of amoebic infection, they examined the centrifuged deposits of 300 pathological urines, but were unable to find any entamoebae.

KHOURI, in five papers (1924 ; 1929 a & b ; 1930 ; 1931), foreshadowed and later fully supported the comments of Deschiens and Melnotte, and severely criticized the whole conception of urinary amoebiasis, with special reference to the workers at Alexandria. In particular he pointed out the strong resemblance which large mononuclear phagocytes may bear to *E. histolytica*, even down to the inclusion of red corpuscles—a point also remarked upon by ALEXEIEFF (1926 and 1927) and by THOMSON (1926)—and stressed that their identity could readily be proved by staining with iron haematoxylin or by cultivation in appropriate media. Commenting on the cases of urinary amoebiasis recorded up to that date he concludes that the condition is extremely rare but has been frequently diagnosed in error by clinicians in cases where no proof was brought forward of the occurrence of amoebae, and that even where indisputable evidence of the occurrence of amoebae was produced, no scientific proof was put forward of the identity of the species or of its pathogenic rôle. Khouri not only examined 50,000 urine samples (over 1 per cent. of which contained blood) in not one of which was he able to detect any *E. histolytica*, but he also made repeated examinations, over a period of several years and invariably with negative results, of the urine of a patient with haematuria, in which his colleagues claimed to have found *E. histolytica*. He asserts that without parasitological proof in the form of wet-fixed, iron-haematoxylin-stained smears showing the characteristic nuclear structure of the parasite, and successful intrarectal inoculation into kittens, the allegation of the occurrence of amoebae in the urine must be regarded as erroneous.

FARMAKIDIS (1930) made similar criticism and emphasized that unremitting search over many years at Alexandria failed to reveal a single genuine case of urinary amoebiasis, despite frequent positive diagnoses by clinicians there.

Notwithstanding these criticisms, records of alleged urinary amoebiasis continued to appear, many of them without the parasitological proofs so justly insisted upon by the authors quoted above. Thus PENSO (1929 and 1932), GAMBIER (1930), VENTURI (1930), SIGNA (1930), GERBASI (1931), BERNASCONI (1931), BRUNELLI (1931), STEFANO (1931), MATTEI (1932) and CROPPER and MITRA (1932) recorded dubious cases. Venturi in particular failed to produce any amoebic infection of a kitten by intrarectal injection of urinary sediment, and although he claimed to have made haematoxylin preparations which showed typical *E. histolytica*, he reproduces only figures of fresh specimens, which are singularly unconvincing. In Gambier's case there appears to have been a possibility that a fistula had formed between the bladder and an amoebic abscess of the rectal wall through which amoebae gained access to the urine. PANAYOTATOU (1930) re-affirmed her belief in the existence of urinary amoebiasis and its frequent occurrence at Alexandria, in flat repudiation of the opinions of Khouri and Farmakidis.

PENSO in 1929 described a case of urinary amoebiasis which, since no amoebae were found in the intestine, he attributes to a new species, *Entamoeba vesicalis*, capable of producing a primary infection of the bladder. He does not, however, state how such a species could gain entry to the urinary tract, and he records the remarkable facts that the amoebae remained alive in urine for a week, actually undergoing multiplication, and that they could not be stained by ordinary methods. Penso redescribed his amoeba in 1932. It seems certain, however, that he was dealing not with a protozoon but with degenerate cells. Nevertheless, many authors subsequently attributed so-called primary cases of urinary amoebiasis to *E. vesicalis*.

DE MELLO (1931) reported one of the few undoubtedly genuine cases of the occurrence of *E. histolytica* in urine. The patient, who was suffering from haematuria and strangury, had no history of previous dysentery but was found to be a carrier. Wet-fixed, haematoxylin-stained smears showed typical active *E. histolytica* in the urine, while some of the urinary deposit inoculated into the rectum of a cat produced a fulminating infection which terminated fatally.

DESCHIENS, in 1933, re-affirmed his previous comments and criticisms in relation to amoebic infection of the urinary tract. However, some further unsatisfactory records of the condition were published by MOSCHINI (1932), GUIDICEANDREA (1932), CAPINCEFF (1934), GIROLAMI (1936), BACHRACH (1936), MANOHAR (1936), KOLLER (1937), and COUTTS and HERRERA (1942). In none of these cases were adequate proofs brought forward, and they can only be regarded as unconvincing.

GIROLAMI's paper, however, includes an exhaustive historical review, a comprehensive bibliography and a critical discussion of the condition; he concludes that urinary amoebiasis is a definite clinical condition which is usually caused by *E. histolytica* and is then secondary to a primary bowel infection, but which may sometimes be a primary infection due to *E. vesicalis* Penso. The figures of *E. vesicalis* which he gives are not convincing. HUARD (1937) follows Girolami's conclusions.

Critical Discussion.

From the preceding historical review it will be clear that in most of the reported cases of infection of the urinary tract with *E. histolytica* satisfactory evidence that the alleged organisms are amoebae is wanting; and even granting that in some cases amoebae may have been present, sufficient proof of their identity with *E. histolytica* is generally conspicuously lacking. In many of these cases, too, the possibility of the alleged amoebae being actually large haematophagous leucocytes or degenerating forms of *Trichomonas vaginalis* has not been definitely excluded. Certain types of leucocytes also bear a close resemblance to *Entamoeba* cysts. Moreover, no evidence is produced in those cases in which amoebae were actually present that they played a pathogenic rôle, the symptoms attributed to their presence being such as are generally found in bacterial and other infections of the kidney, ureter, bladder or urethra in which inflammation has been set up. It will have been noted also that in most of these cases of alleged urinary amoebiasis the final diagnosis was based upon two inadequate premises, namely, the appearance of "amoebae" in the urine examined fresh, and the fact that the condition cleared up after treatment with emetine. As Deschiens and Khouri have pointed out, adequate parasitological proof of the presence of *E. histolytica* is rarely put forward, while the fact that the condition cleared up under emetine therapy cannot be taken as evidence that *E. histolytica* was the causative organism, since the effect of emetine is not confined to specific action upon this amoeba. Indeed, KHOURI (1924) has shown that emetine treatment has a beneficial effect upon bilharzial cystitis, the symptoms of inflammation rapidly disappearing. With these considerations it must also be borne in mind that many of the workers cited above believed that amoebiasis was a generalized blood-borne infection of which amoebic dysentery was merely the most frequent manifestation. Though it is well known that amoebic infection of other parts of the body is always secondary to intestinal infection, yet in many of the recorded cases of urinary amoebiasis no history of previous or concurrent dysentery was given by the patient, and no amoebae could be found in the stools.

However, the fact remains that certain cases, notably those of BAELZ (1883), CRAIG (1911), WALTON (1915), MACFIE (1916), and DE MELLO (1931) were

undoubtedly genuine, and upon the possible nature of these the recent work by WATSON (1945) throws some light. This author tested the ability of *E. histolytica* to live in media contaminated with various proportions of urine and was able to show conclusively that urine at 37°C. is lethal to the parasite, even when diluted to a quarter of its normal concentration. A few cursory tests along similar lines were made by MACFIE (1916), who stressed the harmful action of urine upon *E. histolytica* and noted the similarity of appearance between amoebae from infected faeces which had been immersed in urine and those from the urine of his patient. It is, therefore, doubtful if the parasite can ever exist in a medium of urine, even as dilute as that in the kidney tubules. Watson, however, showed that some time must elapse before the effect of urine upon the amoebae becomes fully evident. Hence, amoebae which gain entry to the lumen of the urethra or ureter, or to the cavity of the bladder, are doomed to extinction unless they can attack the wall of these organs where they will soon become protected from the urine by a crust of necrotic tissue, and may multiply and produce characteristic lesions. Since they retain their active motility for upwards of two hours when immersed in undiluted urine, quite a reasonable chance exists that some of the more vigorous individuals might penetrate the tissue of the urinary tract in this way. The process would obviously be facilitated, as MACFIE (1916) observed, by the prior existence of disease of some part of the urinary tract, resulting either in altered composition of the urine (*e.g.* lower osmotic pressure, lower ammonia and urea content, higher *p*H), rendering it a more suitable medium for *E. histolytica*, or in destruction of the protective epithelium lining the ducts and bladder, and the consequent exposure of areas of the underlying tissue which might be more readily penetrated by the parasite. Presumably, once amoebic lesions become established, active amoebae, together with pus and necrotic tissue, would be extruded into the lumen by compression of the ulcer when the bladder contracts at micturition. The condition in which the amoebae would be found in samples of the urine would depend on how soon, after being passed, the urine is examined. According to WATSON (1945), for a variable period up to about two hours the amoebae are able to retain their active motility, but after a longer interval they become rounded and motionless and gradually degenerate as a result of the influence of the urine upon them.

With regard to the method by which the amoebae gain access to the urinary tract, there are four possible routes, namely :—

(i) Direct access *via* the urinogenital opening and the urethra.

(ii) Entry from the gut into the bladder *via* a fistulous extension of an intestinal lesion.

(iii) Entry into the kidney by extension of a liver abscess.

(iv) Metastatic extension from the intestine *via* the blood or lymphatic channels.

There are thus four distinct conditions in which amoebae might conceivably appear in the urine, in all of which it is evident that a primary infection of the intestine must exist and must serve as the original source of the organisms.

In respect of the first type, unclean habits may lead to the transfer of active amoebae from the rectum and anus to the glans penis or labia and so to the urethral aperture, where, unless rapidly washed away in the course of micturition, they might succeed in penetrating the tissues and setting up a urethritis or an ulcer of the glans itself, as in the cases described by HERMANN and BERMAN (1942), and by SHIH, WU and LIEU (1939). It is obvious that this type of case is liable to continual reinfection. Active amoebae may occur periodically in the urine at irregular intervals.

The second type may occur in individuals in whom the anterior wall of the rectum shows deep and extensive ulceration due to primary amoebiasis, so that perforation of the rectal and bladder walls may occur in the region of the rectovesical connexions, and active amoebae may gain direct access to the bladder. In such cases, which are obviously only likely to occur in male subjects, active amoebae will probably be found frequently in the urine, having passed through into the bladder from the rectal wall. There may be some localized proliferation of the amoebae with consequent ulceration and necrosis of the wall of the bladder in the vicinity of the fistula. The establishment of the fistulous connexions between ulcerations of the gut wall and the bladder is less likely to occur at any other site owing to the freely movable nature of the bladder relative to the intestines at other points, although it is, of course, possible that the development of adhesions elsewhere may facilitate the occurrence. A case of this type was described by CRAIG (1911) in which motile *Entamoeba histolytica* containing red blood corpuscles had been detected in the urine of a patient suffering from acute amoebic dysentery. A few days later the patient died and at autopsy a minute fistula connecting the bladder with an amoebic ulceration of the colon, which was adherent over the bladder, was discovered. Cases which may have been of this type were described by GOYENA (1926), PAWAN (1926) and GAMBIER (1930). A case of this kind is distinctly more likely to occur where amoebic dysentery exists concurrently with vesical schistosomiasis, the bladder wall being already ulcerated by the passage of the helminth eggs. The case described by NOGUE (1922) may have been of this nature.

In respect of the third type, it is well known that an amoebic abscess of the liver may ultimately, unless evacuated by operation, become adherent to, and discharge its contents into, any of the adjacent organs. Rupture into the kidney in these cases, although extremely rare, may sometimes occur, and an amoebic abscess of the kidney may then be formed. Inevitably, amoebae will make their appearance in the urine sooner or later, but are likely to be non-motile and atypical from long exposure to urine. It is evident that infection by this route is more likely to lead to the establishment of the parasite in the urinary system, with resulting ulceration and necrosis, on account of the lower concentration of solutes in the urine of the kidney tubules, than infection leading to the entry of the amoebae into the ureter, bladder or urethra, where the urine is more concentrated. CRAIG (1934) observed in San Francisco a case of amoebic abscess of the kidney, due to perforation of an amoebic abscess of the liver into the kidney, which was discovered at autopsy on a patient who had died of amoebic dysentery. Cases which were possibly of this nature were also described by CARBONARO (1924) and WORSLEY and BATEMAN (1924), but in the absence of direct evidence from operation or autopsy it is unfortunately impossible to be sure.

Recently, two cases of alleged amoebic perinephric abscess have been reported, by KIRSH and DIAZ-RIVERA (1943) and by ROSS (1944). No adequate evidence for the amoebic nature of these lesions, however, is put forward.

With regard to the fourth type, it is known that extension of ulceration in the intestinal wall to include blood vessels permits active amoebae to enter the blood-stream and to be carried by the portal circulation to the liver, and it is claimed that later, when infection is established in this viscus, they may be carried to other organs such as the lung and the brain. In this way the parasite may reach and colonize the organs of the urinary system, setting up characteristic ulcerations. The kidney is the organ most likely to be infected in this way, and the same considerations will then apply as in the third type of urinary amoebiasis, dead and atypical amoebae only being found in the

urine. It must, however, be borne in mind that the possibility of infection of the urinary system in this way is extremely remote, since the majority of the amoebae will tend to be filtered off in the pulmonary circulation or be carried to the brain before they have the chance of reaching the excretory organs.

CRAIG (1934) has laid stress on the large proportion (at least 60 per cent.) of fatal cases of acute or chronic amoebiasis, in which, at post mortem, some form of nephritis is found. He attributes this state of affairs to a peculiar susceptibility of the kidney to toxins which may be elaborated during the progress of the dysenteric infection, and remarks that the importance of direct amoebic infection of the kidney in the production of such lesions is unknown. TRABAUD and MAIRE (1928) had previously reached somewhat similar conclusions as a result of observing nephritic symptoms in several cases of amoebic dysentery.

From the practical point of view, the greatest difficulty in connexion with urinary amoebiasis is the question of diagnosis. The fact that the suspected condition clears up under emetine therapy is not proof positive of its amoebic nature. It must be emphasized that the typical active amoebae showing progressive motility and characteristic hyaline pseudopodia may very likely not be found in the urine unless it has been retained but a short time in the bladder and is examined immediately after it is passed. Non-motile and atypical forms of various types are more likely to occur, especially if the urine is allowed to cool before examination, and these cannot be differentiated from large haematophagous leucocytes unless wet-fixed preparations are made and stained with haematoxylin, when the characteristic structure of the nucleus may be observed. Confirmation of the diagnosis might be obtained by cultivating the amoebae in a suitable medium ; by production of infection in kittens through intrarectal injection ; and by the complement fixation test. Moreover, unless *E. histolytica* can be shown to occur in the intestine, there is practically no possibility that it will be present in the urinary tract, since cysts are only known to hatch in the gut.

Bibliography.

ALEXEIEFF, A. (1926). *Cent. f. Bakt*, v. 101, p. 240.
ALEXEIEFF, A. (1927). *Cent. f. Bakt.* Abt. I, v. 103, p. 354. (See this *Bulletin*, 1928, v. 25, p. 240).
ALEXEIEFF, A. (1927). *Parasitology*, v. 19, p. 333.
ARAVANTINOS, A. & MICHAILIDIS, N. (1918). *Bull. Soc. Path. exot.*, v. 11, p. 685. (See this *Bulletin*, 1919, v. 13, p. 227).
ARLO. (1924). *Marseille Méd.*, p. 1302.
BACHRACH, R. (1936). *Wien. klin. Woch.*, v. 49, p. 1123. (See this *Bulletin*, 1937, v. 34, p. 333).
BAELZ, E. (1883). *Berlin. klin. Woch.*, v. 20, p. 234.
BALSAMELLI, F. (1926). *Cultura Med. Mod.*, p. 497.
BAYMA, T. & FAGUNDES, A. (1918). *Ann. Paulist. Med. Cirurg.* v. 9, p. 173. (See this *Bulletin*, 1919, v. 13, p. 227).
BERNASCONI, F. (1931). *Algérie méd.*, n. 41. [Reference incomplete.]
BRUNELLI, P. (1931). *Arch. ital. Sci. med. colon.* v. 12, p. 218.
BUCHMANN, M. (1925). *Arch. f. Schiffs- u. Tropen-Hyg.* v. 29, p. 342.
CAPINCEFF (1934). *Biologia med.* n. 4. [Reference incomplete.]
CARBONARO, G. (1924). *Policlinico.* Sez. Prat. v. 31, p. 1327. (See this *Bulletin*, 1925, v. 22, p. 11).
CARVAILLO, R. & SAUTET, J. (1926). *Ann. Parasit. humaine et comparée*, v. 4, p. 349. (See this *Bulletin*, 1927, v. 24, p. 18).
CASTELLANI, A. & CHALMERS, A. J. (1919). Manual of Tropical Medicine, 3rd ed., London.
CASTEX, M. R. & GREENWAY, D. (1926). *Bull. et Mém. Soc. méd. Hôpit. de Paris*, v. 50, p. 376. (See this *Bulletin*, 1926, v. 23, p. 641).
CHALMERS, A. J. & O'FARRELL, R. (1917). *J. trop. Med. & Hyg.* v. 20, p. 97. (See this *Bulletin*, 1917, v. 10, p. 137).

CHEREFEDDIN, O. (1927). *Arch. f. Schiffs. u. Tropenhyg.*, v. 31, p. 526. (See this *Bulletin* 1928, v. 25, p. 621).
CHEVALIER, G. & SOULIÉ. (1928). *Rev. prat. Malad. Pays chauds*, v. 8, p. 298.
CHEYSSIAL. (1924). *Marseille méd.* [Reference incomplete.]
COUTTS, W. E. & HERRERA, J. M. (1942). *Med. moderna.* Valparaiso, v. 15, p. 327. (See this *Bulletin*, 1943, v. 40, p. 144).
CRAIG, C. F. (1911). The Parasitic Amoebae of Man. Philadelphia.
CRAIG, C. F. (1934). Amebiasis and Amebic Dysentery. Springfield, Ill.
CRAIG, C. F. & FAUST, E. C. (1943). Clinical Parasitology. London. 3rd edition.
CROPPER, C. F. J. & MITRA, J. (1932). *Indian med. Gaz.*, v. 67, p. 387. (See this *Bulletin*, 1933, v. 30, p. 24).
DE MELLO, F. (1919). *Boll. Ger. Med. e. Farmacia*, v. 5, p. 296. (See this *Bulletin*, 1920, v. 15, p. 192).
DE MELLO, F. (1931). *Arquivos Escola méd.-cirurg. Nova Goa*, No. 6, p. 761. (See this *Bulletin*, 1932, v. 29, p. 255).
DESCHIENS, R. (1933). *Bull. Soc. Path. exot.*, v. 26, p. 178. (See this *Bulletin*, 1933, v. 30, p. 758).
DESCHIENS, R. & MELNOTTE, P. (1928). *Presse méd.*, v. 36, p. 1545. (See this *Bulletin*, 1929, v. 26, p. 465).
DOBELL, C. (1919). The Amoebae living in Man. London.
EVANS, T. C. (1925). *Trans. roy. Soc. trop. Med. & Hyg.*, v. 19, p. 282. (See this *Bulletin*, 1926, v. 23, p. 367).
FANTHAM, H. B. (1916). *Brit. Med. J.*, Apr. 15, p. 553. (See this *Bulletin*, 1916, v. 8, p. 118).
FARMAKIDIS, C. (1930). *Rev. prat. Malad. Pays chauds*, v. 10, p. 508. (See this *Bulletin*, 1931, v. 28, p. 787).
FIESSINGER, N. & PARTURIER, G. (1926). *Bull. et Mém. Soc. méd. Hôpit. de Paris*, v. 50, p. 153. (See this *Bulletin*, 1926, v. 23, p. 373).
FIORITO, G. (1926). *Riforma med.*, v. 42, p. 163. (See this *Bulletin*, 1926, v. 23, p. 371).
FISCHER, W. (1914). *Münch. med. Woch.*, v. 61, p. 473. (See this *Bulletin*, 1914, v. 3, p. 457).
FRANCHINI, G. (1925a). *Pathologica*, v. 17, p. 271. (See this *Bulletin*, 1925, v. 22, p. 738).
FRANCHINI, G. (1925b). *Pathologica*, v. 17, p. 550. (See this *Bulletin*, 1926, v. 23, p. 370).
FRANCHINI, G. (1926). *Riforma med.*, v. 42, p. 843. (See this *Bulletin*, 1927, v. 24, p. 8).
FRIEL, A. R. (1917). *Proc. R. Soc. Med.*, v. 10, Sect. of Path. p. 5. (See this *Bulletin*, 1917, v. 9, p. 425).
GAMBIER, A. (1930). *Bull. Soc. Path. exot.*, v. 23, p. 457. (See this *Bulletin*, 1931, v. 28, p. 264).
GERBASI, M. (1931). *Pediatria*, v. 40, p. 65. (See this *Bulletin*, 1931, v. 28, p. 786).
GIROLAMI, M. (1936). *Arch. ital. Sci. med. colon.*, v. 17, pp. 129 & 228. (See this *Bulletin*, 1937, v. 34, p. 333).
GUIDICEANDREA, V. (1928). *Boll. Accad. med.*, Roma, v. 54, p. 46.
GUIDICEANDREA, V. (1932). *Boll. Accad. med.*, Roma, v. 58, p. 3.
GOYENA, J. R. (1926). *Semana med.*, B. Aires, v. 33, p. 1075.
HERMANN, H. B. & BERMAN, L. S. (1942). *J. Amer. med. Ass.*, v. 120, p. 827. (See this *Bulletin*, 1943, v. 40, p. 457).
HUARD, P. (1937). *Bull. Soc. méd.-chirurg., Indochine*, v. 15, p. 327. (See this *Bulletin*, 1938, v. 35, p. 586).
JACKSON, L. (1922). *J. infect. Dis.*, v. 30, p. 636. (See this *Bulletin*, 1922, v. 19, p. 686).
JÜRGENS. (1892). *Deut. med. Woch.*, v. 18, p. 454.
KARTULIS, S. (1893). *Ztschr. f. hyg. u. Infektionskr.*, v. 13, p. 1.
KHOURI, J. (1924). *Bull. Soc. Path. exot.*, v. 17, p. 811. (See this *Bulletin*, 1925, v. 22, p. 674).
KHOURI, J. (1929a). *Bull. Soc. Path. exot.*, v. 22, p. 158. (See this *Bulletin*, 1929, v. 26, p. 881).
KHOURI, J. (1929b). *Rev. Med. Hyg. trop.*, v. 21, No. 3. (See this *Bulletin*, 1929, v. 26, p. 871).
KHOURI, J. (1930). *Rev. prat. Malad. Pays chauds.*, v. 10, p. 78. (See this *Bulletin*, 1930, v. 27, p. 783).
KHOURI, J. (1931). *Rev. prat. Malad. Pays chauds.*, v. 11, p. 329. (See this *Bulletin*, 1932, v. 29, p. 251).
KIRSH, D. & DIAZ-RIVERA, R. S. (1943). *Amer. J. med. Sci.*, v. 206, p. 372. (See this *Bulletin*, 1944, v. 41, p. 218).
KOLLER, M. (1937). *Arch. f. Schiffs- u. Tropen-Hyg.*, v. 41, p. 593. (See this *Bulletin*, 1937, v. 34, p. 948).
LYNN, W. J. (1914). *Amer. J. trop. Dis.*, v. 2, p. 205. (See this *Bulletin*, 1915, v. 5, p. 200).
MACFIE, J. W. S. (1916). *Ann. trop. Med. Parasit.*, v. 10, p. 291. (See this *Bulletin*, 1917, v. 9, p. 185).
MANOHAR, K. D. (1936). *Brit. med. J.*, Apr. 4, p. 694. (See this *Bulletin*, 1937, v. 34, p. 333).
MANSON-BAHR, P. (1940). Manson's Tropical Diseases. London.

McDill, J. & Musgrave, W. E. (1905). *Med. News*, v. 87, p. 1163.
Mattei, A. (1932). *Ann. Med. nav. colon.*, v. 38, Jan.–Feb., p. 24.
Moschini, S. (1932) *Riv. Clin. pediat.*, v. 30, p. 648.
Nogue. (1919). *Bull. Soc. med-chirurg. française Ouest-Africain*, v. 1, p. 22. (See this *Bulletin*, 1922, v. 19, p. 686).
Panayotatou, A. (1924). *Rev. Med. Hyg. trop.*, v. 16, p. 107. (See this *Bulletin*, 1924, v. 21, p. 683).
Panayotatou, A. (1926a). *Bull. Soc. Path. exot.*, v. 19, p. 88. (See this *Bulletin*, 1926, v. 23, p. 655).
Panayotatou, A. (1926b). *Bull. Soc. Path. exot.*, v. 19, p. 92. (See this *Bulletin*, 1926, v. 23, p. 655).
Panayotatou, A. (1930). *Rev. prat. Malad. Pays chauds.*, v. 10, p. 501. (See this *Bulletin*, 1931, v. 28, p. 787).
Pawan, J. L. (1926). *Ann. trop. Med. & Parasit.*, v. 20, p. 199. (See this *Bulletin*, 1927, v. 24, p. 10).
Penso, G. (1929). *Ann. Med. nav. colon.*, v. 36, Jan.–Feb., p. 32. (See this *Bulletin*, 1931, v. 28, p. 263).
Penso, G. (1932). *Ann. Med. nav. colon.*, v. 38, July–Aug., p. 413. (See this *Bulletin*, 1933, v. 30, p. 24).
Petzetakis, M. (1923a). *Bull. et Mém. Soc. méd. Hôpit. de Paris*, v. 47, p. 1233. (See this *Bulletin*, 1924, v. 21, p. 26).
Petzetakis, M. (1923b). *Bull. et Mém. Soc. méd. Hôpit. de Paris.*, v. 47, p. 1681. (See this *Bulletin*, 1924, v. 21, p. 338).
Petzetakis, M. (1924). *Bull. et Mém. Soc. méd. Hôpit de Paris*, v. 48, p. 1227. (See this *Bulletin*, 1925, v. 22, p. 11).
Petzetakis, M. (1925a). *Rev. Méd. Hyg. Trop.*, v. 17, p. 141. (See this *Bulletin*. 1925, v. 22, p. 738).
Petzetakis, M. (1925b). *Bull. Soc. Path. exot.*, v. 18, p. 478. (See this *Bulletin*, 1926, v. 23, p. 32).
Petzetakis, M. (1925c). *Wien. klin. Woch.*, v. 38, p. 1310. (See this *Bulletin*, 1926, v. 23, p. 370).
Petzetakis, M. & Mylonas (1924). *Bull. Soc. méd. Hôpit., de Lyon.* [Reference incomplete.]
Petzetakis, M. & Petridis, G. (1925). *Rev. Méd. Hyg. trop.*, v. 17, p. 23. (See this *Bulletin*, 1925, v. 22, p. 738).
Peyrot, J. (1924). *Marseille méd.*, p. 1192.
Pirami, C. (1928). *Arch. ital. Sci. med. colon.*, v. 9, p. 143.
Posner, C. (1893). *Berlin. klin. Woch.*, v. 30, p. 674.
Ralli, A. & Panayotatou, A. (1923). *Presse méd.*, v. 31, Aug. 29, p. 742.
Reiss, F. (1924). *Arch. f. Schiffs- u. Tropen-Hyg.*, v. 28, p. 334. (See this *Bulletin*, 1925, v. 22, p. 10).
Rocca, G. C. (1928). *Riforma med.*, v. 44, p. 346. (See this *Bulletin*, 1928, v. 25, p. 622).
Ross, J. A. (1944). *Brit. J. Radiol.*, v. 17, p. 289. (See this *Bulletin*, 1945, v. 42, p. 38).
Rossi, G. (1927). *Riv. Clin. pediat.*, v. 25, p. 433.
Saujeon, M. (1928). *Bull. Soc. Path. exot.*, v. 21, p. 883. (See this *Bulletin*, 1929, v. 26, p. 471).
Shih, H. E., Wu, Y. K. & Lieu, V. T. (1939). *Chinese med. J.*, v. 55, p. 139. (See this *Bulletin*, 1940, v. 37, p. 122).
Signa, A. (1930). *Cultura med. mod.* [Reference incomplete.]
Sitsen, A. E. (1927). *Arch. f. Schiffs- u. Tropen-Hyg.*, v. 31, p. 101. (See this *Bulletin*, 1927, v. 24, p. 777).
Stefano, U. (1931). *Arch. ital. Sci. med. colon.*, v. 12, p. 463.
Stitt, E. R. (1942). Diagnosis, Prevention & Treatment of Tropical Diseases (6th edition, edited by R. P. Strong).
Thomson, J. G. (1926). *Proc. R. Soc. Med.*, v. 19, p. 14. (See this *Bulletin*, 1926, v. 23, p. 859).
Trabaud & Maire. (1928). *Rev. prat. Malad. Pays chauds*, v. 8, p. 357.
van Cutsem Franco, A. (1928). *Bruxelles med.*, v. 8, p. 711.
Venturi, L. C. (1930). *Arch. ital. Sci. med. colon.*, v. 11, p. 465. (See this *Bulletin*, 1931, v. 28, p. 264).
Vichrev, P. (1925). *Arch. f. Schiffs- u. Tropenhyg.*, v. 29, p. 404. (See this *Bulletin*, 1926, v. 23, p. 369).
Viviani, R. (1926). *Morgagni*, v. 68, p. 1089.
Walton, H. J. (1915). *Brit. Med. J.*, May 15, p. 844. (See this *Bulletin*, 1915, v. 6, p. 52).
Ward, A. B., Coles, A. E. & Friel, A. R. (1916). *Brit. med. J.*, Apr. 8, p. 526. (See this *Bulletin*, 1916, v. 8, p. 118).
Watson, J. M. (1945). *Ann. trop. Med. & Parasit.*, v. 39, p. 101.
Wenyon, C. M. (1926). Protozoology. London.
Wijnhoff, J. A. (1895). *Nederl. Tijdschr. v. Geneesk*, v. 31, p. 107.

WORSLEY, A. N. & BATEMAN, J. F. (1924). *J. trop. Med. & Hyg.*, v. 27, p. 278. (See this *Bulletin*, 1925, v. 22, p. 11).
WRIGHT, E. J. (1917). *J. Trop. Med. & Hyg.*, v. 20, p. 145. (See this *Bulletin*, 1917, v. 10, p. 138).
YOKOCHI, K. (1926). *Aichi Igakkwai Zasski*, v. 33, No. 3. (In Japanese). (See this *Bulletin*, 1927, v. 24, p. 365).

MALARIA.

GARNHAM, P. C. C. **Malaria Epidemics at Exceptionally High Altitudes in Kenya.** *Brit. Med. J.* 1945, July 14, 45–7.

In June to August 1941 a severe epidemic of malaria occurred among a small farming community in Londiani (7,500–7,800 feet), 75 miles from the provincial centre, Kisumu, in Kenya. The Londiani district comprises a small township with an Indian bazaar and native location surrounded by European farms within a radius of about 12 miles. There was also a large military camp. Extensive areas of forest reserve border the farms; in these live several thousand African squatters. The malaria epidemic chiefly affected the farms and the military camp; there was but little malaria in the isolated huts of the African squatters. All but one of 27 positive blood slides showed *P. falciparum*; the one exception was *P. vivax*. The epidemic ceased abruptly in August. Smaller outbreaks recurred in each of the three following years at the same season. Many cases were severe.

Seven species of *Anopheles* were found as larvae or adults or both: *A. gambiae*, *A. garnhami*, *A. natalensis*, *A. coustani*, *A. christyi*, *A. squamosus* and *A. kingi*. Of these species only *A. gambiae* is a known vector of malaria. Surveys showed that *A. gambiae* breeds for a short period at these heights about May and that adults persist for the next two months.

In 1944 there were 20 cases of malaria among 220 inhabitants of a farm 20 miles to the north of Londiani at an altitude of 8,300 to 8,500 feet; here two adult *A. gambiae* were found in huts.

A mean temperature of not less than 61°F. has been reported as the limit for *A. gambiae* breeding. This temperature is only attained in four months in Londiani, February to May; the low rainfall of February precludes that month for *A. gambiae* breeding. The mean shade temperature is never sufficiently high in Londiani for the development of the sexual cycle of the parasite in the mosquito, but *A. gambiae* spends most of its life in human habitations where the temperature is from 5 to 10°F. higher than outside; the temperature in huts is just sufficient for the sporogony of *P. falciparum* during the month of May, about 66°F. The survival of *A. gambiae* throughout the year in Londiani is extremely unlikely and it is reasonable to suppose that it is introduced annually by road or rail transport. *A. gambiae* can frequently be caught in railway coaches arriving in Kisumu.

Norman White.

D'ABRERA, V. St. E. **Malaria Parasite Findings in Blood Films in relation to Malaria Forecasts in Ceylon.** *Indian Med. Gaz.* 1945, Feb., v. 80, No. 2, 102–5, 2 graphs.

In this study the author makes use of the relative frequency of the three species of malaria parasite found in blood films from patients attending two dispensaries at Wattegama and Katugastota, in the basin of the Mahaweli Ganga, alt. 1,500 feet, throughout a period of twelve months. Similar observations made by SIVALINGAM from 1938 to 1941 at Giriulla in the basin of Maha

Oya, alt. 200 feet, are also used [see this *Bulletin*, 1943, v. 40, 746]. In these parts of Ceylon, where outbreaks of malaria are influenced by the vagaries of the monsoon, *P. malariae* is most in evidence when the incidence of malaria is low. During seasonal or epidemic rises in malaria, *P. malariae* gives pride of place to *P. falciparum* and *P. vivax*, *P. falciparum* being chiefly responsible for epidemics. An attempt has been made to determine the point in the epidemic cycle at which *P. malariae* begins to give place to *P. falciparum*, and thus to obtain due warning of the approach of an epidemic. The author has found log. M/Q a useful expression where M signifies *P. falciparum* and Q *P. malariae* infections in any given month. The values so obtained are charted. When the curve attains the zero line it is a danger signal. In the three illustrations produced, warnings of approaching outbreaks might have been available two months, two weeks, and one month, respectively, before the event.

Norman White.

DE OLIVEIRA CASTRO, G. M. Ecologia do *Anopheles gambiae*. Pesquisas preliminares sôbre a viabilidade dos ovos que ficam fora da água. [**Ecology of *Anopheles gambiae*. Preliminary Study on the Viability of Ova away from Water.**] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 517–34, 1 graph. English summary (9 lines).

The author, and others [working, apparently, in 1939, before *Anopheles gambiae* was eradicated from Brazil], were surprised by the rapid and intense infestation of the Valley of Banabuiú so soon after the early rains of the year; the valley just before had been apparently dry and free from *A. gambiae*. Ova for these experiments were obtained in the laboratory; they were passed on a piece of moistened filter paper on the top of a cushion of cotton-wool saturated with water in a Petri dish. The laboratory was an emergency one in Aracati, Ceará State.

In the first series the ova were exposed to the ordinary atmosphere away from water, on the filter paper where they had been deposited, the drying thus taking place slowly, within 36 hours. Seven days to a month later they were placed in water. None of the 1,655 ova left for seven days survived to produce larvae, nor did any of 12,276 left for a month. GEBERT had previously reported that ova of *A. gambiae* would not, after four days away from water, give rise to larvae.

Next, the ova were kept away from water, but in air saturated with moisture. They were placed, in lots of 50 or 100, on rectangular pieces of filter paper in small glass tubes with cotton wool (moistened and then squeezed out) at the bottom, and the outlet was closed with a paraffined cork. Some, which were to be kept at a low temperature, were maintained at 14°C. As would be expected, a certain number of the eggs would become larvae and when the time for the test was reached these were counted and rejected, the remainder on the paper being placed in a Petri dish in the centre of which was a pad of cotton wool saturated with moisture, but not enough to cause the ova to come into contact with the water. As controls, other ova were maintained in water at the laboratory temperature. The results were as follows :—

1. *Controls.*—Of 537 ova remaining in the water from the time of oviposition at laboratory temperature, 54 per cent. yielded living larvae, 34·5 per cent. had dead larvae and 11·5 per cent. were sterile.

2. Of those kept *at room temperature away from water* five series of tests were made, estimations being carried out at 8, 10, 15, 20 and 30 days :—

(i) *At 8 days.*—Of 1,149 ova, 10 per cent. yielded living larvae, 70 per cent. died in the tubes, 14·4 per cent. within the ova and 5·6 per cent. were sterile.

(ii) *At* 10 *days.*—Of 1,250 ova 12·4 per cent. gave living larvae, 3 per cent. were sterile and 84·6 had dead larvae (in the tubes or in the eggs).
(iii) *At* 15 *days.*—Of 1,564 kept for this period no living larvae resulted; 2 per cent. were sterile, and 98 per cent. had dead larvae.
(iv) and (v).—*At* 20 *and* 30 *days.*—Readings of these were somewhat spoiled by growth of moulds on the filter paper. The ova were, nevertheless, transferred to water, but no living larvae emerged.

3. Kept *away from water, but at* 14°*C.*
(i) *At* 15 *days.*—Of 1,325 ova there were 5 per cent. with living larvae, 4·2 per cent. sterile and 90·8 per cent. dead.
(ii) *At* 20 *days.*—Of 924 there was 0·1 per cent. living larvae, 23·2 per cent. sterile and 76·7 per cent. dead.
(iii) *At* 30 *days.*—Of 943 ova none yielded living larvae; 9 per cent. were sterile and 91 per cent. dead.

The effect of the sun's rays on pupation was noted. Larvae exposed to the sun pupated in a few hours, even in a few minutes in some instances; when kept in the laboratory away from the sun's rays they did not do so for days.

The conclusion is, therefore, that deprived of water, the ova of *A. gambiae* do not survive for very long. *H. Harold Scott.*

CORRÊA, R. R. & RAMOS, A. S. Notas sôbre o estudo da biologia do *A. tarsimaculatus* em Caraguatatuba. [**Notes on the Study of the Biology of** *A. tarsimaculatus* **in Caraguatatuba.**] *Arquivos de Hig. e Saúde Pública.* São Paulo. 1944, v. 9, No. 20, 103–8. English summary. [Summary taken from *Rev. Applied Entom.* Ser. B. 1945, Aug., v. 33, Pt. 8, 121.]

In view of the finding of malaria parasites in females of *Anopheles emilianus*, Komp (for which the authors use the name *tarsimaculatus*, Goeldi) [see this *Bulletin*, 1942, v. 39, 738] from houses in Caraguatatuba on the coast of São Paulo, in 1943, a study of the bionomics of the larvae and adults of this Anopheline was begun. An account is here given of the methods used.

LEE, David J. & WOODHILL, A. R. **The Anopheline Mosquitoes of the Australasian Region.**

This book is reviewed on p. 1049.

ANDERSON, G. A., VON DESCHWANDEN, J., GRAY, W. A. & MENZIES, T. H. Die Malaria im Hochgebirgsklima. [**Malaria in High-Altitude Climates.**] *Acta Tropica.* Basel. 1945, v. 2, No. 2, 122–36.

This paper deals chiefly with the effects of the climate of Adelboden on patients suffering from malaria, chiefly of the relapsing benign tertian type. Adelboden is a well-known Swiss winter-sports resort situated at a height of about 4,300 feet; snow, frost, and bright sunshine, are the chief features of the climate in winter.

Most of the patients were British soldiers who had fought in North Africa and had escaped from Italian prison camps: they arrived in Adelboden in the months November 1943 to February 1944. There were 84 cases of benign tertian and four of malignant tertian infection; in 34 other cases of clinical malaria no parasites were found. Little information is given of the duration of the infection or of the previous treatment.

Although some of the patients received no specific treatment, hardly any relapses occurred after March 1944; and the striking improvement in their general health was attributed to increased body resistance due to the bracing climate.

Their progress was estimated by systematic examinations of the blood by which remarkable improvement was demonstrated even in cases in which occasional parasitic relapses were still occurring. The blood changes indicating improvement were :—A diminishing blood-sedimentation rate ; an increase in the haemoglobin, red-cell count, and colour index ; a diminution in the number of the monocytes, and in the degree of deviation to the left shown by the Schiller index. So long as these conditions remained abnormal, treatment was regarded as necessary even if no parasites could be found.

John W. D. Megaw.

CHAUDHURI, R. N. & RAI CHAUDHURI, M. N. **Observations on Malaria and other Conditions seen in Burma Evacuees.** *Indian Med. Gaz.* 1945, Feb., v. 80, No. 2, 97–102.

During the exodus from Burma in the six middle months of 1942, 144 evacuees of many races were admitted into the hospital attached to the School of Tropical Medicine, Calcutta. More than half of them had been on the road for six to eight weeks through most unhealthy country, and were in poor shape. This paper is concerned with the diseases from which they suffered. One hundred and five were suffering from malaria, 62 per cent. of them *P. falciparum* infections. Four had typhoid fever complicated by malaria. [See also SCOTT, this *Bulletin*, 1920, v. 15, 252.] There were only two deaths among the 144. Case reports are given of interesting cases together with a mass of miscellaneous information.

Norman White.

HYMAN, A. S. **Clinical Masquerades of Malaria ; Observations in South Pacific Combat Areas.** *U.S. Nav. Med. Bull.* 1945, Aug., v. 45, No. 2, 287–303.

During the first eight months of the Solomon Islands campaign casualties who passed through an advance naval base hospital included a number of cases of malaria diagnosed as such and an equal number of patients suffering from malaria but admitted with other primary diagnoses. A series of 100 such patients admitted with mistaken diagnoses is the basis of this report.

Thirty-four cases had been diagnosed as diseases of the chest, 21 cardiac and 13 pulmonary. The cardiac diseases included angina pectoris, coronary thrombosis, paroxysmal tachycardia, auricular fibrillation, extra systoles and heart block. In all these cases the underlying diagnosis was not arrived at till a chill occurred and malaria parasites were found in the blood. In most patients the cardiovascular symptoms yielded promptly to antimalarial treatment.

The pulmonary diseases diagnosed included atypical or virus pneumonia (8), pleurisy (4), and lung abscess (1). They all responded to antimalarial treatment.

Twenty-eight patients were admitted with symptoms pointing to abdominal disease, nine disease of the stomach, including gastric and duodenal ulcer, eight disease of the gall-bladder or liver or both, six disease of the spleen, and three appendicitis. Illustrative cases are described in detail. Here again the occurrence of a chill and the subsequent discovery of malaria parasites indicated the true nature of the disease.

Twenty-one missed cases of malaria were admitted with diagnoses of bone or joint diseases. In general the picture was one of acute arthritis.

Some disease of the head was the diagnosis in 12 patients, in 7 disease of the brain, 3 of the eyes, 1 of the sinuses, and 1 of the mastoid. Five of the brain cases were admitted with a diagnosis of possible meningitis. Meningococcus meningitis was occurring in minor epidemic form in the South Pacific area at that time; this partly accounted for the difficulty of diagnosis in field conditions.

The 7 remaining patients in this series were admitted with diagnoses of kidney disease, 4; thyroid disease, 2; scrotal disease, 1.

Most of the mistaken diagnoses were made during the early days of the Solomons campaign before most of the medical officers had become acquainted with the bizarre manifestation of South Pacific malaria. In the latter part of the period under review an outbreak of dengue added to the difficulties of diagnosis. *Norman White.*

ARBUSE, D. I. **Neuropsychiatric Manifestations in Malaria.** *U.S. Nav. Med. Bull.* 1945, Aug., v. 45, No. 2, 304–9.

This short paper covers a wider field than its title indicates. Reference is made to the many forms of disease manifestation which may result from the malarial infection of the central or peripheral nervous system. Here the author covers familiar ground. He writes with experience gained in the South Pacific area.

The psychiatric manifestations ascribed to malaria may be personality changes, expressions of the patient's experience of the disease; psychotic symptoms perhaps directly referable to the location of the cerebrovascular thromboses; or a recognizable disease such as schizophrenia or manic depressive psychosis. In many cases transient attacks of depression were noted. In general the psychic symptoms were consistent with the mental make-up of the patients but they may have been activated by malaria. The rapid response and improvement in the mental symptoms with anti-malarial therapy indicates a direct causal relationship in many cases. It is, however, reasonable to suppose that the secondary symptoms such as depressed and paranoid states depend to a considerable extent upon the mental make-up of the patient before he contracted malaria, and that in such cases malaria plays no different rôle from that of other organic diseases which may be followed by psychoses. *Norman White.*

HARVEY, A. M. **A Type of Neuritis associated with Malarial Fever.** *Bull. Johns Hopkins Hosp.* 1944, Oct., v. 75, No. 4, 225–31.

The author reports that in an army general hospital a large group of patients suffering from malaria developed signs and symptoms of peripheral neuritis, which are described. In the mild cases one or more of the extremities "went to sleep" easily, and there were frequent attacks of numbness and tingling. In some cases there was hyperalgesia and hyperaesthesia over the distribution of a peripheral nerve or a posterior root. The symptoms usually increased in severity during subsequent attacks of malarial fever. More severe cases started with sharp pain in the involved region which was followed by sensations of cramp in the muscles. The flexor surfaces of the forearms and hands were usually involved. There was hyperalgesia and increased sweating. The flexion contraction of the muscles in the area could be passively reduced but this caused a great deal of tingling and pain.

These neuritic symptoms may develop during the latent phase of malaria while the patients are taking suppressive treatment. All the patients had mild recurrent attacks of malaria, and all but one were infected with *P. vivax*; the exception had a *P. falciparum* infection. *Norman White.*

KELLNER, A., HOCHSTEIN, E. & TILLMAN, A. J. B. **Spontaneous Rupture of the Spleen in Malaria. Fatal Case.** *J. Amer. Med. Ass.* 1945, Aug. 25, v. 128, No. 17, 1227–9.

RAVDIN, I. S. & NORTH, J. P. **The Simultaneous Occurrence of Acute Appendicitis and Malaria.** *Ann. Surgery.* 1945, Sept., v. 122, No. 3, 432–5.

DAVIDSON, S. **Experimental Investigation of Burasaine in the Treatment of Malaria.** *East African Med. J.* 1945, Mar., v. 22, No. 3, 80–85.

The author reports the treatment with burasaine of 15 non-immune and 3 immune Africans suffering from *P. falciparum* malaria. Burasaine comes from Madagascar. It is the total alcohol extract of the root of a tree, *Burasaia madagascariensis* Thouars (family Menispermaceae). The extract contains alkaloids with the methyl-orthoquinone-isoquinoleic ring such as is found in hydrastine and berberine. The extract was made up into tablets of 5 grains each. Sixteen tablets were given each 24 hours. Swallowing the tablets with water often caused nausea and vomiting, so each dose of four tablets was dissolved in two ounces of glucose water, and 10 minims of dilute hydrochloric acid were given. No toxic effects, except vomiting, were noted. Three non-immune control cases were treated with quinine.

All but one of the patients treated with burasaine only, left hospital free from fever and parasites in the peripheral blood. The resistant case was given quinine on the 19th day and recovered. Burasaine acts very much more slowly than quinine, though from the data supplied it is not possible to make any exact comparison of the efficacy of the two drugs. It would seem that burasaine has some definite antimalarial properties. *Norman White.*

PIRK, L. A. & ENGELBERG, R. **Hypoprothrombinemic Action of Quinine Sulfate.** *J. Amer. Med. Ass.* 1945, Aug. 11, v. 128, No. 15, 1093–5, 2 charts.

The authors have found that the administration of quinine sulphate to normal adults in daily doses as small as 0·3 gm. is accompanied by a significant rise of the prothrombin time, indicating a prothrombin-lowering effect of quinine sulphate. In carrying out the estimations the Page method was used, in which Russell viper venom and calcium chloride are added to plasma obtained by centrifuging decalcified venous blood: the sharp end-point is characterized by the formation of fibrin particles clearly visible in the agitated test tube. If vitamin K be administered with the quinine the fall in the prothrombin level does not occur. The vitamin K compound used was 2-methyl-1, 4-naphtho-hydroquinone-diphosphoric acid ester tetrasodium salt, Synkayvite "Roche." The dose was 10 mgm.

The lowering of the prothrombin level of soldiers taking suppressive quinine would expose them to the danger arising from prolonged bleeding should they be wounded. The concurrent administration of vitamin K is recommended.

Norman White.

DREISBACH, R. H. & HANZLIK, P. J. **Antagonists for the Circulatory Depression of Quinine Injected Intravenously and the implied Cholinergic Action, and Nature and Importance of the Vasodilatation, in the Depression.** *J. Pharm. & Exper. Therap.* 1945, Mar., v. 83, No. 3, 167–84, 3 figs. [16 refs.]

This is a report of an extensive study to discover effective antagonists to the circulatory depression caused by the intravenous injection of quinine. The

experiments were carried out on anaesthetized rabbits and cats ; the technique is described. An adequate summary of the paper is not possible in a small space. Some of the important results obtained are thus summarized by the author :—

" Of the 32 agents tried, three, mixed with the quinine in doses of 10 mgm. per kgm., were capable of preventing or mitigating the depression, namely, epinephrine, 0·015 to 0·02 mgm. per kgm., as the most efficient and desirable, neosynephrine, 0·05 mgm. per kgm., about equal to epinephrine and next in order, and calcium chloride, 10 mgm. per kgm., about equal to epinephrine 0·01 mgm. per kgm., and least efficient and desirable. Thiamine hydrochloride, 10 mgm. per kgm., also antagonized the depression, but the reason for this was not apparent. . . .

" The following groups of agents were found not to be beneficial or desirable : various colloids, ephedrine and benzedrine, calcium in high doses, digitalis group, certain vitamins, a substrate, xanthines, reducing agents, metabolic stimulants, and a number of miscellaneous agents. . . .

" The circulatory depression of quinine was found not to be a cholinergic phenomenon and atropine would be of no antidotal value in mitigating, correcting, or reversing the depressor action, or in collapse from quinine injected intravenously.

" The peripheral vasodilatation of quinine intravenously was found to be an early phenomenon, comparatively minor and evanescent, and of little or no importance in the typical sustained depressor action of the alkaloid. The seat of the vasodilatation was in the peripheral blood vessels, and not demonstrably central, cardiac, or cholinergic in origin."

For " a 70-kgm. adult man . . . the total doses of the most desirable agents for use in severe quinine collapse would be : epinephrine, 1·4 mgm., or neosynephrine, 3·5 mgm., injected intravenously, well diluted and cautiously. Nikethamide, 700 mgm., might be permissible, but would be more hazardous. However, in severe collapse of quinine, the cardiac poisoning may be, and often is, irreversible, and therefore, all treatments may be of no avail."

Norman White.

MACDONALD, G. **Need for Malaria Control Policy for Rural Africa.** *East Africa & Rhodesia.* 1945, Aug. 2, v. 21, No. 1089, 1140–41.

Macdonald reviews briefly the course of malaria in persons born and brought up in conditions of high endemicity such as exist in many parts of Africa. There is good *prima facie* evidence that it causes mortality in infants, and morbidity at later ages, on a large scale, though the statistical evidence for the former is poor. [The findings of the late E. C. SMITH in autopsies on children in Lagos are relevant. Death was attributed to malaria in 14·4 per cent. of a series of 500. See this *Bulletin*, 1943, v. 40, 496.] In spite of the fact that, after a childhood of generally poor health attributable largely to malaria, the adult arrives at a condition of relative immunity, the author cannot agree with those who hold the view that this process should not be disturbed (other than by the administration, from time to time, of such small doses of curative drugs as may suffice to reduce fever) in case the people are deprived of the protection they ultimately achieve through the process of immunity. He quotes findings which indicate that a state of fever is common in childhood and even in adult life, and argues that this morbid process is to the detriment of the people. Most workers would, no doubt, agree with this, the argument of those who hold the view described above would be that at present there is no hope of so reducing malaria in rural Africa that the immune state would be unnecessary, and that partial control would mean loss of immunity, with more

severe reaction to infection, throughout life. Macdonald is not content with this view, and points out that malaria can very largely be controlled in village communities by the spray-killing of adult mosquitoes, and that this procedure is popular with the people. He advocates experiments on a large scale, properly controlled, in which pyrethrum, DDT and gammexane would be used for this purpose, and be combined with control of larvae by hand-strewn paris green. He points out that most of the expense would be the labour, and that any community can afford its own labour. Such experiments should be combined with research work, and the whole project should be conceived on a big scale. At present there is no such large-scale undertaking.

One possible effect of efficient malaria control would be an increase in the size of the populations. This would entail research on the whole subject of agricultural methods and other matters, so that the land could support its people until a balance is achieved at a population considerably above that which now exists. *Charles Wilcocks.*

ANNECKE, A., MCCARTHY, D. D. & WILSON, D. Bagster. [**Dr. G. Macdonald's address on "A Policy of Malaria Control for Rural Africa."**] [Correspondence.] *East African Med. J.* 1945, Sept., v. 22, No. 9, 306–8.

In this letter the writers join issue with MACDONALD [above] over his contention that malaria control should be undertaken on a large scale in the hyperendemic rural areas of Africa, by the combined use of all the means at present available for such control. They argue that, hitherto, eradication has not proved possible in such areas, and that success has been attained, by spray-killing (the only economic procedure) only in places where transmission is seasonal. In places such as Freetown, where circumstances for control by all means are favourable, and in which the population is partially treated and therefore not immune, control should be pushed to eradication, but over the wide hyperendemic areas, where full control measures are not practicable, and where the result of attempts in the past has been merely to reduce the peak of malaria, the resulting diminution of immunity, with prolongation of ill-health throughout life, would more than counterbalance any advantage gained. In such areas "a partial control should not be attempted by present methods, except on an experimental scale"; the writers of the letter urge that such experiment should be vigorously undertaken, so that full advantage may be taken of opportunity for new developments. "What is needed is a new method and one which is much more efficient than any yet known; a single and cheap form of attack is required." To this end they suggest the creation of a Malaria Institute of Africa.

In the meantime, and in view of the fact that malaria is only one of the many health problems of rural Africa, they suggest that creation of better standards of living and agriculture may lead to increased capacity to resist malaria, without losing the essential mechanism of immunity, and would counteract the ill-effects of the present state of under-nutrition.

[A reader may ask how far, in fact, these two points of view actually diverge. The question seems to be limited to hyperendemic areas, since both would probably agree that control is desirable in places where transmission is not perennial, and where immunity is therefore not achieved. Annecke *et al.* argue that in hyperendemic areas, control with present facilities cannot be successful, but that "bonification" (in a wide sense) may increase resistance and retain immunity. Macdonald argues that control may lead to diminution of illness and that bonification may therefore be expected and should be encouraged. Both advocate trial and research, and both recommend agricultural reform. To the reviewer it seems that decision can be reached only through further

experiment, in which each method is given trial under good and thorough conditions; it seems unwise to argue too closely from spray-killing work carried out before DDT was used. As both parties to this argument agree that malaria control should be attempted *on an experimental scale*, in hyperendemic areas, it remains to determine the size of the experiment; *ceteris paribus*, the bigger the experiment, the more clear-cut the results. But it is time this controversy was decided.] *Charles Wilcocks.*

RAINA, B. L. **Larvicidal Fish of Kangra Valley**—*Schizothorax progastus. Indian Med. Gaz.* 1945, May, v. 80, No. 5, 273–5, 1 fig. [10 refs.]

BRUMPT, E. Zooprophylaxie du paludisme. [**Zooprophylaxis in Malaria.**] *Ann. Parasit. Humaine et Comparée.* 1944–1945, v. 20, Nos. 3–4, 191–206. [Bibliography.]

SPECK, J. F. & EVANS, E. A., JR. **The Biochemistry of the Malaria Parasite. II. Glycolysis in Cell-free Preparations of the Malaria Parasite.** *J. Biol. Chem.* 1945, June, v. 159, No. 1, 71–81. [23 refs.] **III. The Effects of Quinine and Atabrine on Glycolysis.** *Ibid.* 83–96. [32 refs.]

II. It was shown by WENDEL [this *Bulletin*, 1943, v. 40, 674] that lactic acid is formed from glucose by *P. knowlesi*. In the first paper of the present series, SILVERMAN *et al.* [this *Bulletin*, 1945, v. 42, 448] showed that it is also formed by *P. gallinaceum*. In the present investigation the mechanism of its formation by *P. gallinaceum* has been studied by using cell-free extracts of the parasite. Two different types of preparation were used, the first was obtained by laking parasitized red cells with water and freeing the parasites from cell debris by centrifugation (haemolysate). It contained enzymes from red cells and parasites and was more active than similar preparations from normal red cells. The second preparation (extract) was obtained by extracting parasites which had been freed from red cells by the saponin method of CHRISTOPHERS & FULTON [this *Bulletin*, 1940, v. 37, 190], or less satisfactorily by laking parasitized cells with water. The operations were carried out at 0°. The results indicated that these enzyme preparations from *P. gallinaceum* were able to phosphorylate glucose in the same manner as yeast extracts, and could convert fructose-1, 6-diphosphate to 3-phosphoglyceraldehyde and bring about the dismutation between the latter and pyruvic acid. [FULTON (this *Bulletin*, 1940, v. 37, 509) found by manometric methods that *P. knowlesi* was unable to oxidize hexose di- or mono-phosphate.] The cell-free extracts of *P. gallinaceum* appear to convert glucose to lactic acid in a manner similar to that of other living cells.

III. Quinine and atabrine were shown by SILVERMAN *et al.* (see above) to inhibit glycolysis and oxygen uptake by *P. gallinaceum*. By investigating the effect of these drugs on the phosphorylation of glucose and the dehydrogenation of 3-phosphoglyceraldehyde and lactic acid by the enzyme preparations used above, it was sought to throw light on their mode of action in malaria. Experiments of a similar nature were made at the same time with glycolytic enzymes, from yeast and muscle. It was found that quinine in concentrations of from 0·0001 to 0·002 M inhibited the phosphorylation of glucose in haemolysates of normal chicken red cells, but was much less active against similar preparations from parasitized cells. The inhibition corresponded to the amount of hexokinase—the enzyme which catalyses the phosphorylation—obtained from red cell material. In the case of atabrine, at similar concentrations to those of quinine, phosphorylation of glucose was inhibited in both haemolysates to the same extent. Each drug inhibited the action of yeast hexokinase but the

enzyme 3-phosphoglyceraldehyde dehydrogenase, which is concerned in lactate formation, was not significantly inhibited. Lactic dehydrogenase in the cell-free preparations from *P. gallinaceum*, as well as in ox-heart preparations was more effectively inhibited by atabrine than by quinine. The latter drug consistently inhibited glycolysis by muscle extract, and also phosphorylation of glycogen, as well as the reverse reaction of polysaccharide formation from phosphorylated glucose, besides other enzyme reactions involved in sugar-phosphorus exchange. Considered in conjunction with the observations of SILVERMAN *et al.* on the inhibition of glycolysis by quinine and atabrine in the case of *P. gallinaceum*, the present results suggest that these two drugs may interfere with the hexokinase and lactic dehydrogenase of the parasites. Similar results were obtained on the inhibition of glycolytic enzymes from other systems. The authors point out, however, that the drug concentrations employed by them are greater than those which obtain during therapy. It seems to them improbable that interference with the glycolytic mechanism of the parasites is of chief importance in the therapeutic action of these drugs, but rather that their oxidative processes, especially those concerned with lactic and pyruvic acid, are interfered with. *J. D. Fulton.*

PARAENSE, W. L. O ciclo exoeritrocitário dos parasitos da malária. [**The Exo-Erythrocytic Cycle of Malaria Parasites.**] *Mem. Inst. Oswaldo Cruz.* 1944, Dec., v. 41, No. 3, 469–93, 3 pls., 7 figs. [Numerous refs.]

BLACKWATER FEVER.

MAEGRAITH, B. G., HAVARD, R. E. & PARSONS, D. S. **Renal Syndrome of Wide Distribution induced possibly by Renal Anoxia.** *Lancet.* 1945, Sept. 8, 293–6. [65 refs.]

The authors critically discuss the question of what is the direct or immediate cause of the pathological changes in the kidneys which are present in persons in whom, usually during an acute illness, renal failure (with or without oliguria or anuria) occurs.

" The onset of oliguria or anuria is commonly the first sign. In patients who survive there is also a postanuric period of impaired renal function with nitrogen retention and often a copious unconcentrated urine." This condition of the kidneys is distinguished from other forms of nephritis chiefly by the fact that the kidneys recover completely in surviving patients.

The macroscopical and microscopical appearance of the kidneys in fatal cases is described in detail and reference is made to various pathological states in which similar renal changes occur (" larval " or " necrotising " nephrosis, and " prerenal azotaemia " of FISHBERG ; " tubulo-vascular renal syndrome " of MAEGRAITH).

Anuria in many different diseases, *e.g.* blackwater fever and cholera and in trauma in man and animals (in experiments) is discussed with reference to the views and suggestions of various authors. Three causes of the renal changes have been suggested : (1) Mechanical blockage of the kidney tubules ; (2) the action of some injurious substance in the blood ; (3) renal anoxia. The authors discuss these fully and conclude that the condition of the kidneys is probably caused by renal anoxia, usually secondary to peripheral vascular failure. [See also TOMB, this *Bulletin*, 1942, v. 39, 163, 459, in relation to cholera, and HILL, *Bulletin of War Medicine*, 1941, No. 6, 355, with regard to crush injuries.] *J. F. Corson.*

TRYPANOSOMIASIS.

HUNT, A. R. & BLOSS, J. F. E. **Tsetse Fly Control and Sleeping Sickness in the Sudan.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1945, Sept., v. 39, No. 1, 43–58, 2 text figs., 4 figs. on 1 pl. & 1 graph. [13 refs.]

Sleeping sickness due to *Trypanosoma gambiense* has been endemic in parts of the southern Sudan for many years, and has never been eliminated from the area round Tembura, adjoining French Equatorial Africa. In this area there are many small rivers, tributaries of the Yubu, almost all of which are infested with *Glossina palpalis fuscipes*. *Glossina morsitans* also exists in the area, but apparently is not connected with human infection ; it follows game, and the inhabitants are persistent hunters whose activities tend to drive away the animals, and with them *G. morsitans*.

Attempts have previously been made to control sleeping sickness in this region by the creation of special settlements along roads, and by control of traffic, with some, but never complete, success. As the disease was reduced, however, control measures were neglected ; many of the regulations were irksome to the people. The authors have not found *G. palpalis* more than 10 yards from water unless carried away by the host, and in 1938, therefore, they began the block method of control described by SYMES and his collaborators [this *Bulletin*, 1936, v. 33, 651 ; 1937, v. 34, 542 ; 1939, v. 36, 740].

The fly density in the Yubu area was small (average 9 to 12 per fly-boy-day) compared with that reported by Symes from Kenya (168 per fly-boy-day) and the blocks made by the authors were therefore considerably larger than those of Symes ; barrier clearings were made 800 yards long and 400 yards wide. The maintenance of clearings was difficult, but various cover crops were planted, to replace the rapidly growing local spear grass ; of these the most successful was colopogonium. As the scheme progressed and the disease incidence fell, the maintenance of small road-river crossing clearings and of watering place clearings was discontinued, and the people were asked to maintain the barrier clearings as a civic duty ; this entailed less than one-fifth of the work they previously did.

In the six blocks prepared, in which fly-boy paths were cut as close to the water as possible, catching was done by teams of boys aged 11–16 ; the total staff now numbers six group leaders, 26 section leaders and 220 fly boys, who patrol 300 miles of paths.

The results are shown in a series of diagrams, covering the past five years. These indicate very clearly the great reductions that have been made in the numbers of flies caught ; a catch as low as five or less per fly-boy-week had been attained over most of the area by 1943, and this rate has been maintained. Complete elimination of the fly is, apparently, not possible by these means.

In the Yubu area there were 65 cases of sleeping sickness in 1938, but only three in 1942, though in other areas outside the scope of this control the numbers had not diminished [these figures presumably refer to new infections diagnosed]. The disease, therefore, has very largely been controlled, in spite of the fact that many irksome restrictions have been removed.

Charles Wilcocks.

BLOSS, J. F. E. **The Meridi Outbreak of Sleeping Sickness.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1945, Sept., v. 39, No. 1, 59–76, 1 map, 2 diagrams & 5 figs. on 1 pl.

In February 1941 an epidemic of sleeping sickness broke out near Meridi in Equatoria Province of the Sudan ; no indigenous case had previously been

reported from this area, though *Glossina palpalis* was fairly common along the Eidi river, and *G. morsitans* in the more open country to the north. The trypanosome concerned was *T. gambiense*. The outbreak was very localized, and to control it the block method of Symes was introduced, a census made, and inspections of the population held every three months. By the end of 1941 a total of 47 cases had been found, in 1942 there were 25 new cases, in 1943 there were nine. Details are given of the construction of blocks and clearings, which were similar to those used during the Yubu outbreak [see HUNT and BLOSS, above] except that at a later stage narrow (50 yards) rod clearings were substituted for the wider (150 yards) clearings at first made, in two places. One feature was the use of bamboo and thatch screens, one of which was erected at each end of a clearing. The screens were placed across the river and extended for 10 yards up each bank, and each was reinforced by two secondary screens further out from the river and overlapping the first; these were known as staggered screens. These screens acted as barriers against flies which might otherwise have entered or crossed the clearings, since GIBBINS [this *Bulletin*, 1942, v. 39, 442] has shown that such obstructions will deter the flies from travelling along the course of a stream. These were successful in improving the efficiency of barrier clearings.

The author remarks that on the subject of control of *G. palpalis* there are now two views; one favours the block method, with continued fly catching and wide clearings between blocks, the other favours rod clearings, relatively narrow but often of great length, assisted by screens, and controlled by periodic catches. Each probably has its place, and further work is to be done to test the methods.

Diagnosis rested on the old method of gland palpation, by which up to 1,000 people can be tested in one day, followed, in suspected cases, by gland puncture and blood examination after triple centrifugation. This is efficient to detect an outbreak provided that enough of the people are seen. The fly population of this area was low—never over 30 per fly-boy-week—and it seems that the flies do not migrate far from people, and that they tend to infect those who use the same watering place. The disease can, therefore, establish itself when fly density is low, and fly control must, in consequence, be thorough and effective; a "safe" low density would be under five flies per fly-boy-week with continuous catching.

The incubation period was long; cases were rarely found within six months of infection, and it may be a year before the disease is recognizable. Germanin (or antrypol) and tryparsamide were used in treatment, but the author found that a course of six injections of germanin was insufficient for first-stage cases and advises that all should be treated with the same full course (3 germanin followed by 5–10 tryparsamide) as is given in second- or third-stage cases.

Charles Wilcocks.

ERRATA.

In the abstract of Dr. A. F. FOWLER's paper in this *Bulletin*, 1945, v. 42, 541, the sentence "Trypanosomes were present in the cerebrospinal fluid in 33 of the 42 patients" should read, as explained by the author in his paper, "Positive C.S.F. means that there was either direct or indirect evidence of central nervous system involvement. Thus thirty-three cases, *i.e.* 78 per cent., showedinvolvement of the central nervous system". The abstracter regrets to have overlooked this explanation of the + sign in the table.

In the title of the paper by EAGLE abstracted in this *Bulletin*, 1945, v. 42, 354, the Greek letter *gamma* was misprinted as V; the name of the drug should read γ-(p-Arsenosophenyl)-Butyric Acid.

HAWKING, F. **The Absorption of 4 : 4′-Diamidino Stilbene (Stilbamidine) by Trypanosomes and its Blood Concentration in Animals.** *J. Pharm. & Exper. Therap.* 1944, Sept., v. 82, No. 1, 31–41, 4 figs. [12 refs.]

It was shown by HAWKING [this *Bulletin*, 1939, v. 36, 220] that trivalent arsenicals and acriflavine are rapidly absorbed by trypanosomes *in vitro*. The absorption of 4 : 4′ diamidino stilbene (stilbamidine) by trypanosomes previously demonstrated by HAWKING and SMILES [this *Bulletin*, 1942, v. 39, 238] with the aid of the fluorescent microscope has been further investigated as a guide to the mechanism of its trypanocidal action. Some determinations of the plasma levels of the drug were also made by the method of HENRY and GRINDLEY [this *Bulletin*, 1943, v. 40, 122], in mice and rabbits at various times after injection. It was found that living trypanosomes absorb a large amount of stilbamidine at 37°C., but in contrast to the author's experience with acriflavine and trivalent arsenicals, dead trypanosomes do not absorb it in any appreciable amount. The value of the partition ratio of the drug (concentration in trypanosomes/concentration in surrounding fluid) diminished with increasing concentration of the drug over the range studied. The ratio was smaller than that for trivalent arsenicals or acriflavine, indicating a less marked absorption of stilbamidine by the trypanosomes (*T. equiperdum* and *T. rhodesiense*). Stilbamidine is also absorbed more slowly than the other drugs and the amount taken up at 18°C. is negligible. The absorption process was shown to be reversible in the early stages. The quantitative estimation of the amount of stilbamidine in trypanosomes isolated from the blood of a treated animal provided evidence of its absorption *in vivo* by these parasites additional to that previously obtained by the fluorescent microscope. The drug disappeared rapidly from the plasma of treated animals. From the above results the amount required to kill a single trypanosome was estimated. In general, the action of stilbamidine on trypanosomes closely resembled that of the other drugs mentioned, but the site of absorption on the parasite is probably different as judged by the facts of drug resistance [this *Bulletin*, 1939, v. 36, 221]. *J. D. Fulton.*

KING, H. **Chemical Structure of Arsenicals and Drug Resistance of Trypanosomes.** Reprinted from *Trans. Faraday Soc.* No. 265. 1943, Dec., v. 39, Pt. 12, 383–7. [17 refs.]

The work of EHRLICH and others on drug resistance in trypanosomes is briefly reviewed. In regard to the mechanism of drug resistance, YORKE, MURGATROYD and HAWKING [this *Bulletin*, 1931, v. 28, 909] first showed by means of a biological method that normal trypanosomes removed reduced tryparsamide from the medium, whereas drug-fast trypanosomes failed to do so. This fact was confirmed by HAWKING (*J. Pharm & Exper. Therap.*, 1937, v. 59, 123) by chemical means. Arsenophenylglycine and phenylarsenoxide were, however, absorbed by normal and resistant trypanosomes. GOUGH and KING (*J. Chem. Soc.*, 1930, 669) observed that a series of aromatic arsonic acids, in which certain acidic groups were present, were non-curative in experimental trypanosomiasis, whereas the corresponding amides had curative properties. Later it was found that a number of arsenicals containing carboxyl groups, in which the –As : As– linkage was present, also possessed trypanocidal properties. Phenylarsenoxide, as Hawking had found, as well as derivatives with or without carboxyl groups, proved lethal to normal and arsenic-resistant strains of trypanosomes *in vitro*. From these facts it was concluded that arsenicals act primarily on trypanosomes in at least three different ways. The substances containing carboxyl groups form readily soluble, highly ionized, neutral sodium salts and the ions do not readily leave the water phase, a fact which may explain their

low trypanocidal power. One of the substances studied was 4-phenyl glycine arsenoxide and it is suggested that the anomalous behaviour of the phenylglycine derivatives is due to the presence of the carboxyl group.

Non-ionized substances probably act on trypanosomes in a different way. Some arsenoxides without marked polar or hydrophilic groups are very active against normal and resistant trypanosomes *in vitro*. Since certain trypanosomes are known to contain large amounts of lipoid material, the author suggests that the active arsenoxides orient themselves at a lipoid-water interface, so that the phenyl or corresponding group is in the lipoid and the arsenoxide group is at the water phase, whereby they are easily mobilized at the site of action on the trypanosome. Judging by the activity of certain members against both normal and resistant strains, they act in a different way from the other substances studied. These latter types, by which drug-fastness can be produced, are possibly adsorbed on polar surfaces. The action of phenyl -*pp'*-diarsenoxide on normal and resistant trypanosomes was found to be similar to that of atoxyl, and the author therefore concludes that both ends of such molecules are involved in the primary fixation on the trypanosome. The final reaction by which the trypanosome is killed probably involves the arsenoxide group and the SH group in the living cell. *J. D. Fulton.*

ROMAÑA, C. & MEYER, H. Estudo do ciclo evolutivo do "Schizotrypanum Cruzi" em cultura de tecidos de embrião de galinha. [**Study of the Developmental Cycle of** *T. cruzi* **in Fowl Embryo Tissue Culture.**] *Mem. Inst. Oswaldo Cruz.* 1942, v. 37, No. 1, 19–27, 12 figs. on 6 pls. English summary.

"Chick embryo tissue cultures were smeared with *Schizotrypanum* from different sources. The cultures were inoculated with flagellates from blood-agar cultures and in one instance from blood of an infected guineapig.

"Carrel's technique of tissue culture, with slight modifications, was employed.

"The tissues used were spleen, myocardium, liver, epithelium of the iris, spinal ganglion and monocytes from chicken blood.

"In all these tissues the flagellate developed easily, parasitizing different types of cells: fibroblasts, histiocytes, macrophages, epithelial cells, cells of the nervous system, etc.

"The authors were able to follow the complete cycle of the parasite, observing its classic evolution: transformation into leishmania, multiplication by binary division, transformation into crithidia and finally into trypanosome.

"They also observed forms of the parasite which possibly developed directly from leishmania to trypanosome without apparently going through the crithidia stage.

"They succeeded in infecting a white mouse with a human strain of *S. cruzi* after passage through tissue culture.

"The authors also pointed out different phenomena observed in the relations between the cells and the parasites."

NERY GUIMARÃES, F. & JANSEN, G. Novo transmissor silvestre do "*Trypanosoma* (*Schizotrypanum*) *cruzi*" (Chagas, 1909). [**A New (Natural) Transmitter of** *Trypanosoma cruzi*.] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 437–41, 3 text figs. & 3 figs. on 1 pl. English summary.

In October 1942 the authors were carrying out research on American trypanosomiasis in the Santa Teresa district and found the opossum, *Didelphis*

aurita, infected in nature and the transmitting insect, *Panstrongylus megistus*, similarly infected. In the course of their researches the authors found another bug, only one-third to one-fourth the dimensions of the *P. megistus*, naturally infected, in the nests of the opossum in the branches or stems of the palm, *Attalea indaya*. Sixteen specimens of nymphs and adults of this insect were examined and seven were found infected with the trypanosome. Infection was proved by inoculation of the rectal contents intraperitoneally into guineapigs and the subsequent infection of the latter by xenodiagnostic methods 25 days later. The new transmitter is said to be closely allied to the genera *Belminus* and *Bolbodera*. *H. Harold Scott.*

LENT, H. Novo transmissor da doença de Chagas na cidade do Rio de Janeiro, D.F. Estudo dos gêneros *Belminus* Stal, 1859, *Bolbodera* Valdés, 1910 e descrição de *Parabelminus carioca* n.g., n.sp. (*Hemiptera, Triatomidae*). [**A New Transmitter of Chagas's Disease in the Town of Rio de Janeiro,** *Parabelminus carioca.*] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 497–516, 10 figs. [32 refs.]

The author has made a careful examination of the new transmitter of *T. cruzi* discovered by GUIMARÃES and JANSEN when carrying out investigations in the Santa Teresa district (see above). In the report of the latter it was stated that the insect appeared to be related to the genus *Belminus* or to *Bolbodera*. In the present article, the author gives an account of the characters of each of these genera and shows that the new insect belongs to neither, but to a new genus which he calls *Parabelminus* ; he names the type species *carioca*. The affinities between the three genera are shown in a table and a very detailed account of the new vector is given in the letterpress, with drawings of the various parts. The original should be consulted by entomologists and others interested as the details are too minute to be transcribed here [they occupy four pages of small print]. [See previous paper for comparison in size between it and *Panstrongylus megistus*]. *H. Harold Scott.*

LENT, H. & DE OLIVEIRA, S. J. Nota preliminar sobre a ação do DDT (dicloro-difenil-tricloroetana) em insetos transmissores da doença de Chagas. [**Preliminary Note on the Action of DDT on the Insect Vectors of Chagas's Disease.**] *Rev. Brasileira Biol.* 1944, v. 4, No. 3, 329–31. [Summary taken from *Rev. Applied Entom.* Ser. B. 1945, Aug., v. 33, Pt. 8, 132.]

An account is given of preliminary laboratory experiments carried out in Brazil with a view to using DDT [2, 2-bis (parachlorphenyl-1, 1, 1-trichlorethane] for the control of Triatomid bugs. In the first test, *Rhodnius prolixus*, Stål, *Panstrongylus megistus*, Burm., *Triatoma sordida*, Stål, and *T. infestans*, Klug, were placed on petri dishes dusted with Neocide (5 per cent. DDT adsorbed on talc). The action of the DDT was slow, particularly on the immature stages, the later nymphal instars being the most resistant. One fifth-instar nymph of *T. sordida* moulted after 10 days' exposure to DDT, giving rise to an adult that became paralysed. Most of the bugs began to show signs of being affected after exposure for 18–24 hours, but survived, lying on their backs, for several days. Pure, crystalline DDT did not appear to act more quickly than Neocide. DDT did not impede embryonic development ; eggs of *R. prolixus* hatched after being left in a treated dish for 20 days.

In a second series of experiments, 0·07–0·5 gm. pure DDT in gelatinous capsules was administered to pigeons weighing about 350 gm. on which individuals of *R. prolixus* were later allowed to engorge. The results were somewhat contradictory. With doses of 0·07, 0·2, 0·3–0·4 and 0·45–0·5 gm., the pigeons' blood was toxic to the bugs 3–6, 12–20, 1½–2½ and 1½–27 hours, respectively, after the birds had ingested the DDT. The pigeons were not affected by doses of 0·4 gm. or less, but those that received 0·45–0·5 gm. were affected seriously and sometimes fatally, and bugs that fed on them 1½–6 hours after they had ingested the DDT were affected within 1½–3 hours.

ROMAÑA, Cecilio & DIAS, E. Reação de fixação do complemento na doença de Chagas, com antígeno alcoólico de cultura do "Schizotrypanum cruzi". [**Complement Fixation in Chagas's Disease, with Cultures of *T. cruzi* as Antigen.**] *Mem. Inst. Oswaldo Cruz.* 1942, v. 37, No. 1, 1–10. [13 refs.]

For preparing their antigen the authors grow the trypanosome in a medium of glucose broth and rabbit blood, haemolysed by distilled water. The details are as follows :—

In a Roux flask is placed 100 cc. of nutrient agar ; after this is sterilized and cooled to 50–55°C., 10 per cent. of rabbit (or sheep) blood is added and after being shaken is left to solidify. The surface is then covered with 100 cc. of glucose nutrient broth, *p*H 7·0–7·2. The medium is then seeded with 10 cc. of the trypanosome-containing material (from man, opossum or bat) and kept at 20–25°C. for 15 to 20 days.

The fluid portion of the culture is then drawn off ; the deposit is centrifuged and washed three times with saline. The fluid is again drained off and to the deposit is added ten times its volume of acetone and the mixture shaken repeatedly during the next 24 hours. After centrifugation, the deposit is placed in the incubator at 37°C. to dry and the product weighed. To this, absolute alcohol is added in the proportion of 1 cc. to each centigramme of the powder. This is kept in a well-stoppered bottle for 20 days at 37° being frequently shaken. It is then fit for use.

Of 27 cases of Chagas's disease the sera of 26 gave a positive result ; one was negative, although xenodiagnosis was positive. Of 12 chronic cases with myocardial signs, goitre and "mal de engasgo" [see this *Bulletin*, 1921, v. 18, 301 ; 1927, v. 24, 830] seven were positive. Of nine cases of cutaneous leishmaniasis seven were positive and "the causative organism of this is closely allied to *T. cruzi*," but in cases of syphilis, malaria, ankylostomiasis and other diseases, and 14 normal sera, all failed to give a positive reaction. One patient with yaws gave a positive, but in this case there was a strong suspicion that he was suffering also from Chagas's disease. *H. Harold Scott.*

ROMAÑA, C. Contribuição ao conhecimento da patogenia da Tripanosomose Americana. (Periódo inicial da infecção.) [**Pathogeny of the Initial Period of Infection in Chagas's Disease.**] *Mem. Inst. Oswaldo Cruz.* 1943, Dec., v. 39, No. 3, 253–64, 18 figs.

For studying the histology of the early stage of infection by *T. cruzi* the author scraped the inner aspect of the thighs of white rats and deposited thereon some drops of the metacyclic cultural forms of the trypanosome. Animals were killed and examined at varying intervals afterwards. One killed on the fifth day showed leishmania and intracellular trypanosomes in the corium, subcutaneous cellular tissue and the subjacent muscle, but no cellular infiltration around the parasitized cells. There was a sparse infiltration with polymorphonuclears, eosinophiles, monocytes, some lymphocytes and histiocytes about the broken nests of parasites, and leishmania could be

seen in the phagocytes. The one killed on the 7th day showed greater numbers of parasitized cells; there were no reactionary phenomena round the whole cells, but infiltrative foci were more abundant, and migratory histiocytes were numerous, some enclosing the protozoa. On the 11th day the phenomena described were more marked—the cellular infiltration of the corium, the subcutaneous tissue and the subjacent muscle. The inflammation was specially marked in the fatty tissue where many of the cells were parasitized. In the infiltrated area polymorphonuclears, monocytes and histiocytes containing whole or partly destroyed leishmania were visible. The muscles showed similar changes, their fibres showing parasites with no surrounding reaction, except that those destroyed or broken were invaded by migrating phagocytes. There was also interstitial oedema of the corium and subcutaneous cellular tissue. The inflammatory phenomena were ascribable to the trauma of the raspatory, not to the parasites. In brief, during the first period, that of intracellular multiplication of the parasites, infiltrative cellular phenomena do not occur; these are not seen till 4 or 5 days afterwards, in consequence of rupture of the parasitized cells.

Without scarification, that is, mere contact does not bring about infection, or very rarely. The nasal mucosa is a site easy to infect, the buccal mucosa less so. *H. Harold Scott.*

Couceiro, A. Lesões do ciático na infecção experimental de cães pelo *Schizotrypanum cruzi*. (Nota prévia.) [**Lesions of the Sciatic Nerve in Experimental Infection of Dogs with** *T. cruzi*.] *Mem. Inst. Oswaldo Cruz.* 1943, Dec., v. 39, No. 3, 435–9, 12 figs. English summary (4 lines).

Villela and Torres have described a paralysis occurring as a result of the experimental infection of dogs with *T. cruzi*, but, apparently, no minute examination was made of the peripheral nerves, and the present author believes that these played a part in causing the symptoms. Villela and Torres ascribed the symptoms to central lesions and described them [see this *Bulletin*, 1926, v. 23, 63; 1927, v. 24, 593]. The author apologises for the small number of his observations, the findings in four dogs (six were the subjects of experiment but two died of intercurrent disease), but what he has to record is quite definite.

Macroscopically, the sciatic nerves showed no obvious inflammation or signs of degeneration, but, microscopically, there was visible a perivascular infiltration of the vessels in the nerve-sheaths, with proliferation of the endothelium and perithelium, and in both these leishmania were seen, so that some of the capillaries seemed to be obliterated by them; in others the leishmania decreased from the loose connective tissue cells to those of the neural sheath. In the cells of the perineurium they were distributed longitudinally, or in rounded masses; the myelin sheaths appeared in some places to be swollen and to stain poorly, whereas others were normal. Though there is not evidence enough to show that *T. cruzi* has any true neurotropism, the histological picture, as regards the nerves, is that of a neuritis, predominantly interstitial. *H. Harold Scott.*

Muniz, J. & de Freitas, G. Contribuição para o diagnóstico da doença de Chagas pelas reações de imunidade. I. Estudo comparativo entre as reações de aglutinação e de fixação do complemento. [**Immunity Reactions in the Diagnosis of Chagas's Disease. I. Comparison between Agglutination and Fixation of Complement.**] *Mem. Inst. Oswaldo Cruz.* 1944, Oct., v. 41, No. 2, 303–33, 1 fig. [16 refs.] English summary.

The primary difficulty in carrying out this research was to find a satisfactory antigen for the complement fixation test. Those of historical interest

were all tested, from that of GUERREIRO and MACHADO in 1913 to that of DAVIS in 1943 [see this *Bulletin*, 1943, v. 40, 890]. The *pros* and *cons* of each are given, but suffice it that the most recent one, that of Davis, proved the best, since it was a potent antigen, kept well, and was not anticomplementary to any appreciable degree. The details for its preparation are given [and are recorded in this *Bulletin*, *loc. cit.*]. The sera tested numbered 54 altogether; of these 6 were from acute cases of Chagas's disease, 16 from chronic cases, 7 from suspected. All these 29 were human patients; in addition there were 4 animal sera, one of a dog infected with *T. cruzi*, 2 of armadillos suspected of this infection, and one of a horse with *T. equinum*. There were also sera from 11 persons suffering from other diseases: malaria (3), yaws (3), leprosy (4), cutaneous leishmaniasis (1), and from ten normal subjects.

Twenty-four of the Chagas's disease patients were diagnosed by the finding of parasites in their blood or by xenodiagnostic methods, and all except one gave a positive complement fixation reaction. This one was positive to xenodiagnosis; three others were positive to the complement fixation test but negative with xenodiagnosis. The leprosy, malaria and yaws patients gave negative tests; the one with cutaneous leishmaniasis was very weakly positive.

As regards the agglutination test, all but one of the Chagas's disease patients gave a positive in a serum dilution of 1 : 160 to 1 : 10,240, the acute cases reacting in higher dilution (from 1 : 2,560 upwards) than the chronic (from 1 : 160 to 1 : 2,560). Those suffering from other diseases, malaria and cutaneous leishmaniasis, were negative, but some normal sera and others from leprosy and yaws cases, and the horse with *T. equinum*, agglutinated in low dilution, none higher than 1 : 80. The two reactions, agglutination and complement fixation did not run *pari passu*; one might agglutinate strongly but fix complement only in high concentration. The authors conclude :—

"Until further data on these reactions will be available, complement fixation test is the most secure method for the diagnosis of this disease and the authors believe that with the introduction of Davis antigen and of methods for mass culture of the flagellates in accordance with the technic herein given, this test is perfectly apt to be included amongst routine methods for laboratory diagnosis of Chagas disease." *H. Harold Scott.*

LEISHMANIASIS.

ANGEVINE, D. M., HAMILTON, T. R., WALLACE, F. G. & HAZARD, J. B. **Lymph Nodes in Leishmaniasis. Report on 2 Cases.** *Amer. J. Med. Sci.* 1945, July, v. 210, No. 1, 33–8, 2 figs.

The two cases reported were in young soldiers of the U.S. Army who were admitted to hospital in England early in 1944 for enlargement of lymphatic glands, particularly those of the cervical region. In both cases sections of glands removed by biopsy revealed leishmania. In one case a sternal puncture failed to show any parasites; in the second case sternal puncture was not performed. In both there was complete freedom from other symptoms or signs. Both were treated with sodium antimony gluconate solution, one receiving a single dose of 6 cc. daily for 15 days and the other two such doses daily for seven days. It appeared that the infection in both cases had been

contracted in North Africa or Sicily during the latter part of 1943. Sections of the excised glands showed a moderate fibrosis of the capsule, and the cortical lymph spaces filled with mononuclear cells and lymphocytes. The lymph follicles were relatively indistinct, with considerable hyperplasia of the central parts. There was also a hyperplasia of the reticular network of the node. Throughout the pulp were focal areas of decreased cellularity consisting of a spongy oedematous stroma enclosing scattered histiocytes. Leishmania were found most frequently in such sites, where there was a tendency to the formation of multinuclear giant cells in which parasites were present. Lymph nodes removed later showed that the spongy areas were infiltrated with and replaced by fairly compact sheets of pink-staining epithelioid cells. In lymph nodes showing this reaction it was not possible to demonstrate parasites. It is thought that the histological features of the lymph nodes in these leishmania infections should be of assistance in establishing a diagnosis and separating them from other chronic granulomatous conditions. It seems that the first of the two cases described in this paper is the one referred to in a paper by BURCHENAL and WOODS (this *Bulletin*, 1945, v. 42, 635). *C. M. Wenyon.*

SENEKJIE, H. A. & LEWIS, Ruth A. **Diagnosis of Leishmaniasis by Slide Agglutination.** *Proc. Soc. Exper. Biol. & Med.* 1944, Oct., v. 57, No. 1, 17–19. [17 refs.]

Sera were obtained by immunizing rabbits with 10 injections of 0·5 to 1·0 cc. of killed cultures of *Leishmania brasiliensis*, *L. donovani*, *L. tropica* and *Trypanosoma cruzi* (this *Bulletin*, 1943, v. 40, 681). After sera had been collected from the animals they were still further immunized by repeated injections of living cultures. From the flagellates, slide agglutination antigen and "H" and "O" antigens were prepared. The various sera were tested against these three antigens. It was found that on the slide the homologous organism was agglutinated to a high titre, but agglutination to a lower titre was obtained with the heterologous organisms. In all the flagellates tested "H" and "O" agglutinogens were present. It seems possible that a rapid diagnosis of visceral, cutaneous and muco-cutaneous leishmaniasis could be made by slide agglutination. *C. M. Wenyon.*

MACKAY-DICK, J. **Comments on Kala-Azar with a Report on Three Cases.** *J. Roy. Army Med. Corps.* 1945, Aug., v. 85, No. 2, 68–74.

HARDY, J. D. & PASSMORE, R. **Kala-Azar as seen in an Indian Base General Hospital.** *J. Indian Army Med. Corps.* 1945, July, v. 1, No. 2, 26–8.

BATTELLI, C., COCLANI, A. & ROSSI, M. Ricerche sulla diffusione della leishmaniosi del Cane in Eritrea. **Incidence of Leishmaniasis in Dogs in Eritrea.** *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1944, v. 4, No. 3, 497–505. [17 refs.] English summary (5 lines).

The authors have examined smears of the spleen and bone-marrow of 86 dogs in Asmara, Eritrea, for evidence of leishmania infection. Parasites were discovered in eight of the animals. Various serological tests were also carried out. These were positive (+ + + or + +) in most of the infected dogs but a small percentage of the apparently healthy animals gave a similar result. The various findings are set out in tabular form. Canine kala azar was previously recorded from Eritrea by CONTI in a dog in Ghinda (*Boll. Soc. Ital. Med. Col.*, 1931, p. 170). *C. M. Wenyon.*

FULTON, J. D. **Penicillin in Leishmania Infections.** [Correspondence.] *Nature.* 1945, Aug. 18, 203.

The author has treated hamsters experimentally infected with the parasites of Indian kala azar. They were treated daily over a period of eight days, two with doses of 400 Oxford units administered intraperitoneally or subcutaneously every 3 or 4 hours, and two with half this amount. There was no evidence that penicillin had any influence on the course of the infection. Tests were also carried out on the flagellate forms of leishmania in cultures. Here again penicillin, even in high concentration, was without any lethal action on the flagellates. *C. M. Wenyon.*

FERNÁNDEZ CASTANYS, A. Nuevas aportaciones al tratamiento del kala-azar infantil por el hexonato de antimonio en solución oleosa y concentrada. [**Oily Suspension of Hexonate of Antimony in Treatment of Infantile Kala Azar.**] *Rev. Sanidad e Hig. Pública.* 1945, Feb., v. 19, No. 2, 114–24, 5 figs. on 1 pl.

In an earlier communication (*Actualidad Médica,* July 1943) the author reported on the successful treatment of two cases of infantile kala azar in Spain with the suspension in oil of the hexonate of antimony (sodium antimony gluconate) which was referred to in a paper by KIKUTH & SCHMIDT [this *Bulletin,* 1944, v. 41, 194]. In the present paper he reports on the treatment of seven other cases in children varying in age from 7 months to 3 years. The total quantity administered intramuscularly in each case varied from 10 to 19 cc. This quantity was given in 10 daily injections or in 7 injections on alternate days. In none of the cases was there any evidence of toxicity. The injections were well tolerated, and response, as shown by cessation of fever, reduction in the size of the spleen, and improvement in the general health, was rapid. There appeared to be every reason for assuming that a cure had been effected in all the seven cases. The author concludes that treatment with the oily suspension is the method of choice for cases of infantile kala azar.

The author at the end of his paper makes some remarks on the epidemiology of kala azar. He notes that Dr. MORENO of Granada, in a long experience of the disease, has observed that in every case seen by him there has been a history of a previous case in the same house. It would seem that a reservoir of infection must exist. As in some cases there has been no association with dogs, cats or fowls, these animals cannot be implicated. It is suggested that the lizards (*Platydactylus mauritanus*) may become infected by feeding on mosquitoes which have bitten the children. The mosquitoes again may convey infection from the reservoir lizards to the children. No experimental evidence in favour of or against this speculation is produced. *C. M. Wenyon.*

FAROOQ, M. & QUTUBUDDIN, M. **Oriental Sore in the Nizam's Dominions : some Epidemiological Factors.** *Indian Med. Gaz.* 1945, Feb., v. 80, No. 2, 85–9, 1 map. [10 refs.]

Oriental sore having been reported from time to time from Jalna and Pattan, in the Aurangabad district in the most northern part of the Hyderabad Province, the authors thought it worth while to make a careful survey of the population in these two towns as well as in Aurangabad. The populations of the three towns are :—Aurangabad 50,924, Jalna 22,408, and Pattan 6,294. The incidence per thousand of oriental sore, in most cases diagnosed by the

discovery of leishmania in the lesions, in the samples of the population examined (1,032, 3,242 and 1,781) was 4·84, 27·76 and 42·68. The majority of cases were in children below the age of 10. Females appeared to be as commonly infected as males. Sandflies were most prevalent in Pattan, where sanitation was very poor. The commonest species was *Phlebotomus papatasi*, of which two specimens—one from Jalna and one from Pattan—showed flagellate infection of the midgut. *P. argentipes* was also taken in fair numbers, while only a single specimen of *P. sergenti* was captured, in Pattan. Three species or varieties of the *minutus* group—*P. antennatus*, *P. babu* and *P. bailyi*—were also represented. *C. M. Wenyon.*

LOPES, C. F. & LAENDER, J. F. A intradermo-reação de Montenegro no diagnóstico da leishmaniose tegumentar Americana. Empregos de antigenos velhos—intradermo-reação negativa em casos de bouba. [**Montenegro Skin Test in Muco-Cutaneous Leishmaniasis. Negative for Yaws.**] *Brasil-Medico.* 1945, Feb. 3 & 10, v. 59, Nos. 5 & 6, 41–6. [10 refs.] English summary.

The authors carried out the Montenegro skin test in 50 cases of muco-cutaneous leishmaniasis in Brazil, employing as antigen a suspension in saline of killed culture flagellates of *Leishmania brasiliensis*. A reaction of at least 72 hours' duration and of varying intensity was obtained in 92 per cent. of the cases, 48 of which were examined for the presence in the lesions of leishmania, which were demonstrated in 24. In four cases there was a negative result in spite of the fact that leishmania were discovered in all. The four cases, however, were of cutaneous lesions only, of not more than 6 months' duration. In two of the cases, a father and daughter, the test was repeated twice with the same result. The reactions in cases with lesions of the mucous membranes were generally much stronger than in cases with cutaneous lesions only. The reaction was also more marked in cases of long duration. It was also positive in two cases which had been cured one and three years before. Tests were carried out with two samples of an antigen which had been stored in the ice box for one and four years. Both were found to be quite satisfactory, a finding which is of importance to those who wish to keep stocks of antigen in more remote places. As controls, 17 cases of other diseases including five cases of yaws were tested. All of these gave negative results. It is concluded that the Montenegro test, as maintained by a number of earlier observers whose work is briefly reviewed, is the method of choice for the diagnosis of muco-cutaneous leishmaniasis in the clinics and health centres of the hinterland. *C. M. Wenyon.*

FEVERS OF THE TYPHUS GROUP.

PROC. ROY. SOC. MED. 1945, July, v. 38, No. 9, 511–18 (Sect. Comp. Med. 21–8), 1 fig. [28 refs.] **Discussion on the Control of Rickettsial Infections** [STUART-HARRIS, C. H.; CARMICHAEL, J.; LEWTHWAITE, R.; PARISH, H. J. (President); FULTON, F.; FELIX, A.; FOX, L. A.; LONGLEY, E. O.; HOLE, N.].

This discussion, held on April 18, 1945, was opened by Lt. Col. C. H. STUART-HARRIS, who reviewed the whole subject of the prevention of typhus infection by delousing, early diagnosis, isolation, and immunization by vaccines.

DDT was used for delousing; great help was obtained from a rapid bedside slide test in which stained cultures of *Proteus OX19* were employed. Since 1943 the vaccine used in the British Army has been prepared from yolk-sac suspensions purified by being extracted with ether according to the method introduced by Craigie in 1942.

The Naples epidemic, which in December 1943 looked like assuming disastrous proportions, was rapidly brought under control by searching for infected persons and dusting every possible "contact" with a 10 per cent. DDT powder. The Allied-Army Medical Officers, Red-Cross workers, and civilian personnel, were helped by workers of the International Health Division of the Rockefeller Foundation and of the American Typhus Commission; at the height of the campaign as many as 60,000 persons were being deloused every day.

Dr. J. Carmichael referred to the tick-borne rickettsial infection called heart-water disease which occurs among cattle, goats and sheep, in South Africa; he said that the most effective method of prevention was by repeated arsenical dips for the destruction of the ticks. He also said that "tick typhus" in dogs might be of importance because of its possible transmission to man, but that it was easily controlled by applying derris, pyrethrum, or arsenical washes to the dogs.

Mr. E. O. Longley described a fatal disease occurring among dogs in Nigeria; he thought that this was probably rickettsial, and he mentioned that he had suffered from an attack of typhus following a bite by a tick which was believed to have come from a dog. He added that previous cases of typhus fever in West Africa had been regarded as murine in origin, "probably on insufficient evidence." [Dogs and other domestic animals, even when they are susceptible to rickettsial infection, are not necessarily important reservoirs of the infection: they may serve chiefly as conveyers of ticks infected from wild animals.]

Dr. R. Lewthwaite gave a clear summary of modern knowledge of scrub typhus which, in his opinion, ought to be called tsutsugamushi. He stressed the close relationship existing between this disease and the louse-borne, flea-borne, and tick-borne typhus fevers; "only the most expert pathologist could distinguish between the morbid anatomy and morbid histology of louse-borne and mite-borne typhus." His comments on mite-repellents, mite-poisons, and vaccine prophylaxis, were necessarily uninformative owing to the censorship still imposed on publication of the methods by which the disease was being successfully controlled. Flame-throwers were mentioned as having been used with success in "civilizing" camp sites. Neither chemotherapy nor serotherapy had met with success. Many lives had been saved by nursing the patients "in situ" instead of sending them on a fatiguing journey to hospital. An example was given of the immunity that results from an attack of the disease; among 28 workers deputed to graft palm trees in a highly infected area there were 7 who had previously been patients in hospital and had been proved to be cases of scrub-typhus; all these remained healthy, whereas 10 of the remaining 21 workers were attacked.

Dr. Forrest Fulton said that when vaccines were standardized by counting the rickettsiae, it was found that yolk-sac and animal-lung vaccines gave similar degrees of protection. He suggested that the antigenic relationship between typhus rickettsiae and *Proteus OX19* was heterologous.

Dr. A. Felix suggested that serotherapy might be made more effective by adding anti-*Proteus OX19* serum to anti-rickettsial serum because the latter is known to contain little of the heat-stable antigen which is an important element in producing immunity.

Brigadier General Leon A. Fox of the U.S.A. Typhus Commisssion said that he knew of no authenticated case in which a person vaccinated with the

Cox-Craigie vaccine had afterwards died of typhus fever. He believed that when a large proportion of a population was vaccinated an epidemic could not get started, and that an application of DDT rendered a person non-infestable for 3–4 weeks. *John W. D. Megaw.*

JETTMAR, H. M. & SHIGAN LI. **Weil-Felix Reaction of Men and Rats in Kweiyang.** *Chinese Med. J.* Washington. 1944, July-Sept., v. 62, No. 3, 227–30.

In Kweiyang, where a mild form of typhus fever occurs [see WU & CHI, this *Bulletin*, 1945, v. 42, 795] the authors carried out agglutination tests on 352 sera sent for the Wassermann tests and on the sera of 349 rats. The reactions obtained are shown as percentages in the table :—

Titre	Human Sera			Rat Sera		
	1–40	1–80	1–160	1–40	1–80	1–160 to 1–1,280
Proteus OX19	28·1	6·3	1·4	2·3	1·7	1·5
Pr. OX2 ...	5·1	1·7	0·6	3·2	0·6	0·6
Pr. OXK ...	11·4	4·8	1·1	10·9	4·9	2·4
Bact. typhosum	47·2	21·0	6·8	Not done		

Pregnant rats showed a much higher percentage of positives than the others ; very young rats were seldom positive. In 37 rats *Trypanosoma lewisi* were found, and in 18, *Grahamella muris* ; in both of these groups a high percentage gave positive reactions against *Proteus* strains.

[No other figures are given in connexion with these interesting findings.]

Rats immunized against *Pr. OX19* showed a considerable degree of para-agglutination for *OXK* but very little for *OX2*. Rats immunized against *OXK* agglutinated *OX19* almost as strongly as *OXK*.

The authors state that, clinically, the disease in Kweiyang does not suggest the severe classical type, so it " may be considered to be of the murine type."

[The severity of louse-borne typhus is so variable that it is a very unreliable differential diagnostic feature.] *John W. D. Megaw.*

COHEN, S. S. **The Chemical Alteration of a Bacterial Surface, with special reference to the Agglutination of** *B. proteus* **OX-19.** *J. Exper. Med.* 1945, Aug. 1, v. 82, No. 2, 133–42, 4 figs. [15 refs.]

The author has employed microelectrophoresis in the investigation of the agglutinins concerned in the Weil-Felix reaction. He has applied this method in studying the effect produced by the addition of benzene sulphonyl chloride to cultures of *Proteus OX19* and has presented a number of hypotheses as possible explanations of the effects observed.

The work is of a highly technical nature and will be of interest only to investigators into the antigenic structure of organisms used in agglutination tests. The author points out that *Pr. OX19* has at least two antigenic groups, one of which reacts with agglutinins contained in typhus serum, the other with agglutinins produced in *Proteus* infections, not necessarily of the *OX19* type, so that the Weil-Felix reaction is not absolutely diagnostic of either of these infections.

Even with rickettsia-agglutination reactions the differential diagnosis of epidemic and murine typhus is complicated by the complex nature of the antigenic structure of the rickettsiae. *John W. D. Megaw.*

FITZPATRICK, Florence K. **Studies on Rickettsial Agglutination in Typhus.** *J. Lab. & Clin. Med.* 1945, July, v. 30, No. 7, 577–86. [22 refs.]

Most of the rickettsial suspensions used in these investigations were prepared by thoroughly shaking collections of infected yolk sacs in bottles containing glass beads, adding formolized (0·2 per cent.) normal saline to make a 20 per cent. suspension which was kept at 2–5°C. for 3–7 days and was shaken daily; the suspension was filtered through gauze and then extracted with an equal volume of ether in the cold; about 18 hours later the antigen was drawn off and phenyl mercuric nitrate was added to make a concentration of 1–50,000.

Serum dilutions were made in tubes; a measured drop of each dilution was placed separately on one of 12 hollows on a glass slide and an equal drop of rickettsial suspension was added to each drop of diluted serum. The slide was placed on moist paper in a Petri dish and kept for 5 hours in an incubator at 40°C.; it was then kept overnight in an ice-box. The results were read with a low-power microscope (×100).

The following abbreviations are used.

M-R-A=Murine rickettsial agglutination. E-R-A=Epidemic rickettsial agglutination; R-M-S-F-R-A=Rocky Mountain spotted fever rickettsial agglutination; and W-F=Weil-Felix reaction (*Proteus OX19*).

Numerous sera from healthy persons were examined, but no positive reactions occurred with any of the rickettsial suspensions. Among 155 sera from persons with a clinical history of typhus fever [presumably murine] within the previous two years 104 were positive with the M-R-A test at titres ranging from 1–20 to 1–1,280. In 58 of the 104 positives the W-F reaction was negative or at a lower titre; in 18, the W-F titres were higher than the M-R-A.

In two cases of laboratory infection with epidemic typhus in previously vaccinated persons the W-F, E-R-A, and M-R-A titres began to rise on the 5th day in one case, and on the 8th day in the other; in the former case the maximum E-R-A and M-R-A titres (1–160) were the same; in the latter the E-R-A titre was 1–640 and the M-R-A titre was 1–160.

Among the sera of more than ninety persons recently inoculated with epidemic-typhus vaccine of varying potency only 6 showed E-R-A titres as low as 1–20 and only one was completely negative; all the sera yielding these poor responses were from persons who had received vaccine of low potency. The rest were positive in higher titre. The W-F responses were insignificant in all the cases in which comparison could be made with the pre-vaccination titres.

Among sera from 23 naturally infected rats, 15 were positive with the M-R-A test and though most of these also reacted with the E-R-A test the titres were always lower; only one of the sera was positive with the W-F test.

Guineapigs inoculated with epidemic or murine rickettsiae showed a rapid rise in titre when tested with the homologous rickettsiae; often also when tested with heterologous rickettsiae, but usually at much lower titres. In one lot of 6 mice inoculated intraperitoneally with murine yolk-sac rickettsiae, M-R-A titres of + to +++ 1–40 were reached by all within a week, and 4 of the mice had similar titres for the E-R-A test. Another group of 6 mice inoculated intravenously with epidemic rickettsiae were completely negative with the M-R-A test and all reacted with the E-R-A test. Mice inoculated intraperitoneally with epidemic rickettsiae showed some cross agglutination for murine rickettsiae, but always at a much lower titre than for epidemic rickettsiae, so that there was evidence of a very distinct antigenic difference between the epidemic and murine strains.

Absorption tests were found useful in the differentiation of the strains. After repeated absorption of the sera of murine typhus patients by *Proteus*

antigen it was found that the rickettsial agglutinins were still intact, but after absorption with murine rickettsiae both the *Proteus* and murine agglutinins were removed.

Sera from 40 murine-typhus patients, 20 of which were tested singly and 20 others in one pool, were completely negative with the R-M-S-F-R-A test, and sera of rabbits which had been hyperimmunized with Rocky Mountain spotted fever rickettsiae did not agglutinate typhus rickettsiae. The rickettsia-agglutination reactions in these diseases appear, therefore, to be fully specific. The author remarks that this finding is opposed to the results reported by PLOTZ *et al.* [this *Bulletin*, 1945, v. 42, 636] who found that sera from 13 of 14 cases of Rocky Mountain spotted fever agglutinated typhus rickettsiae. The report by Plotz is also said to be at variance with "unpublished observations in this laboratory on the sera of spotted fever cases, and is therefore puzzling."

The test is regarded as a simple method of detecting typhus infection in a rat population and as being particularly useful in the diagnosis of mild cases of human typhus. Murine and epidemic strains can be differentiated from each other and from Rocky Mountain spotted fever. The test may also be the only available means of diagnosing mild or inapparent infections in guineapigs.

[The discordant results obtained by leading experts in connexion with the Rickettsia-agglutination reaction in cases of Rocky Mountain spotted fever will cause disappointment to those who hope for a simple and truly specific test by which to differentiate between the various fevers of the typhus group. The author's findings might even encourage clinicians to dream that one day they may be provided with sets of standardized rickettsial suspensions by which rapid slide tests could be employed for this purpose. This dream might still come true if it should turn out that the author's method of preparing the suspensions has eliminated a disturbing factor responsible for the occurrence of non-specific agglutination.] Presumably Plotz used rickettsial suspensions purified by repeated washing and centrifugation. According to WERTMAN [see this *Bulletin*, 1945, v. 42, 635] relatively crude yolk-sac suspensions contain appreciable quantities of non-specific antigen capable of causing false positives in the highly sensitive complement-fixation technique employed by Plotz, who therefore purified his suspensions as stated above. The ether-purification method introduced by CRAIGIE for the preparation of improved typhus vaccines [see STUART-HARRIS, above] may turn out to be more suitable for the production of highly specific suspensions for the rickettsia-agglutination tests. *John W. D. Megaw.*

BENGTSON, Ida A. **Applications of the Complement-Fixation Test in the Study of Rickettsial Diseases.** *Amer. J. Pub. Health.* 1945, July, v. 35, No. 7, 701–7.

The author summarizes and brings up to date the results of her very extensive work on the complement-fixation test as a method of diagnosis in Rocky Mountain spotted fever, endemic and epidemic typhus fevers.

Among 216 sera giving positive fixation for Rocky Mountain fever, 92·1 per cent. had no cross fixation for endemic-typhus antigen; 3·7 per cent. gave slight cross fixation; and 4·1 per cent. gave cross fixation at titres ranging from 1–32 to 1–256, but these titres were always much lower than those obtained with Rocky-Mountain antigen.

Among 114 sera positive for endemic typhus, 80 per cent. gave no cross fixation for Rocky Mountain fever; 9·6 per cent. gave slight cross fixation; and 9·6 per cent. gave cross fixation at titres of 1–16 to 1–11,024; here again the cross-fixation titres were much lower.

Among 322 sera tested also by the Weil-Felix reaction, 23·9 per cent. were positive with the complement-fixation test and negative with the Weil-Felix test: this finding occurred in 49 cases of Rocky Mountain spotted fever and in 28 of endemic typhus. In certain cases there was a positive Weil-Felix reaction and a negative complement fixation, but in these cases a later fixation test usually gave "more definite results," and in cases of persistent divergence the possibility of *Proteus* infection has to be considered.

The long persistence of the complement-fixation reaction is a valuable feature, it often facilitates a retrospective diagnosis. The test was also found very valuable in detecting infection among rats; among 1,392 sera of rats from areas where endemic typhus occurred, 46·7 per cent. were positive with the fixation test and only 3·6 per cent. with the Weil-Felix test. The fixation test is highly specific; several hundred normal sera, 150 sera of atypical-pneumonia patients, and 40 sera found positive for tularaemia, undulant fever, or typhoid fever, were negative for endemic typhus. Reactions at titres of 1–4 can, therefore, be regarded as significant.

When specific antigens become generally available, say in the form of vaccines, the test may become a routine procedure, complementary to the Weil-Felix test. *John W. D. Megaw.*

LEÓN, A. P. Fijación del complemento por el suero de enfermos de tifo exantemático al *Proteus* OX–19. [**A Complement-Fixation Test for Exanthematic Typhus, using** *Proteus OX19.*] *Rev. Inst. Salubridad y Enfermedades Trop.* Mexico. 1945, Mar., v. 6, No. 1, 15–30, 2 graphs. English summary.

Using an antigen prepared by a special technique from cultures of *Proteus OX19*, the author has tested the sera of 94 patients by the complement-fixation method; the titres in 84 cases were 1–80 to 1–81,920, whereas by the Weil-Felix test 79 of the patients reacted at titres of 1–80 or over.

In 238 healthy controls the reaction was negative in 92 per cent., and the highest titre observed was 1–160. From one of the tables it appears that among the sera of 37 typhoid patients three were positive 1–320, four showed a titre of 1–160 and two a titre of 1–80.

The reaction was found more sensitive than the Weil-Felix test, especially during the early days of the illness. There was a close degree of correlation between the titres of fixation and the Weil-Felix titres.

Contradictory findings by other workers are attributed to differences in the methods of preparing the antigen; after failures with other methods the author hit on a technique which he describes in detail; he insists that this must be followed exactly if good results are to be obtained. The cultures were suspended in distilled water, concentrated by centrifugation, resuspended in distilled water, shaken in a vessel containing glass balls, and preserved by the addition of phenol and sodium chloride.

A curious finding was that the sera of 14 healthy rabbits reacted at titres of 1–120 to 1–600 and that no increase in these titres resulted from inoculation with *Rickettsia prowazeki*. Among guineapigs found negative before inoculation with *R. prowazeki* 60 per cent. became positive afterwards.

John W. D. Megaw.

DE LA CAMARA, J. P. Observaciones sobre un brote de tifus exantemático en Madridejos (Toledo). [**Observations on an Outbreak of Exanthematic Typhus Fever in Madridejos (Toledo).**] *Rev. Sanidad e Hig. Pública.* 1944, Dec., v. 18, No. 9, 653–60, 2 graphs.

In this outbreak 43 persons were attacked between November 1942 and April 1943. The source of infection was not discovered, and as the first case

was atypical, infection had already spread before the nature of the disease was recognized.

In 34 of the cases, contact with another patient was known to have occurred within the limits of the incubation period, but in several cases the latest contact had been a month or more previously ; the author assumes that an " inapparent" attack must have intervened to maintain the chain of infection. Less than half of the attacks were typical, the others were mild and might easily have been mistaken for influenza, especially as no rash could be detected. All the attacks in children under 15 years of age were atypical, and one inapparent case was detected by the occurrence of a positive Weil-Felix reaction.

Sera from 38 patients were tested ; of these, 31 reacted with *Proteus OX19*, 33 with *Pr. OX2*, and 33 with a strain supplied as *Pr. OXK*, but found to be different. All the sera reacted with one or more of these strains ; 27 reacted with all three, and the average titre with *OX2* was higher than that with *OX19*.

[These findings, like many others, suggest that the agglutinating properties of the strains used by different workers in carrying out the Weil-Felix test are liable to considerable variations. There is a great need for standardized suspensions and uniformity in technique if the findings of different observers are to be comparable.] *John W. D. Megaw.*

GIROUD, P. & PANTHIER, R. Comportement du rat à l'inoculation péritonéale de virus historique passé par lapin (souche pulmonaire lapin). [**The Response of the Rat to Peritoneal Inoculation of Historic Virus passed through the Rabbit. (Rabbit-Lung Strain).**] *Ann. Inst. Pasteur.* 1945, July–Aug., v. 70, Nos. 7–8, 248–9.

The authors have already shown [this *Bulletin*, 1945, v. 42, 104] that historic strains of typhus rickettsiae might behave either as orchitic or non-orchitic strains according to the conditions in which they were transmitted by intraperitoneal inoculation through guineapigs. When suspensions of infected rabbit lung were used to initiate the transfers through guineapigs, and further passages were made with mixtures of vaginal exudate and spleen substance, orchitis was produced up to the eighth passage, but when brain substance was used no orchitis was produced.

In the present experiments three strains of historic rickettsiae which had been carried through 60 or more rabbit-lung passages were used in attempts to transmit infection through rats by intraperitoneal or lung inoculation. There was complete failure to adapt any of the strains to passage through rats even when massive doses of rabbit lung substance were used to initiate the passages, and even when the passages were initiated by vaginal exudate obtained from guineapigs of the fourth or fifth intraperitoneal passage—the initial guineapig passage having been made with infected lung substance of rabbits at the 80th or later lung passages. Mouse-lung substance was used in one experiment ; a mouse at the 88th lung passage was the source of the infected material ; the result was the same as when rabbit lung was used.

The authors conclude that " the rat is a sure test for the differentiation of the historic virus and the murine virus."

From the details supplied by the authors it appears that a few of the rats primarily inoculated intraperitoneally with massive doses of infected lung substance died within three days ; some rats yielded a few rickettsiae from the vaginal exudate, but in most cases no rickettsiae were found, and the infection could not be maintained by further passages.

In the attempts to carry out lung passages through rats, febrile reactions occurred—in one case up to the fifth passage—but no rickettsiae could be detected after the initial passage. *John W. D. Megaw.*

DE MAGALHÃES, O. & ROCHA, A. Contribuição para o conhecimento do tifo exantematico neotrópico no Brasil. [**Contribution to the Study of Typhus in Brazil.**] *Mem. Inst. Oswaldo Cruz.* 1944, Feb., v. 40, No. 1, 1–8.

The authors describe the effects of the inoculation intraperitoneally into certain wild animals of suspensions of tissue of the central nervous system of guineapigs infected with a strain (V.B.) of Brazilian exanthematic typhus ; the animals used were the coati (*Nasua narica*), a species of wild cat (*Felis wiedi*), a species of ferret (*Orison vittatus*) and a species of armadillo (*Tatus novemcinctus*). These animals got an inapparent infection ; their blood gave positive Weil–Felix reactions and the presence of circulating virus was demonstrated by inoculation of their blood into guineapigs, which became infected.

Some points in regard to Brazilian exanthematic typhus in man are mentioned : fever is often absent, and the same applies to infections of susceptible animals whether with the active virus VB or the attenuated viruses VA1 and VA2. Splenomegaly is a much more important symptom of the infection than fever. The authors classify the clinical forms of the disease in man into inapparent, benign, grave, and fulminating types ; the grave type is further subdivided into the common grave form, the nervous or meningo-encephalic form, and the anuric form. They have seen very similar petechial rashes in Brazilian exanthematic typhus and typhoid fever and think that this similarity has led to confusion in the past. *J. F. Corson.*

BERGER, E. & BRZEZINSKI, S. Die Inaktivierung des Fleckfiebervirus durch Organextrakte und Fettsäuren. [**Inactivation of the Virus of Typhus Fever by Extracts of Organs and by Fatty Acids.**] *Schweiz. med. Woch.* 1945, June 23, v. 75, No. 25, 568–70. [11 refs.]

The authors have demonstrated that pronounced antitoxic and antirickettsial effects are produced in mouse-lung suspensions of murine and classic typhus rickettsiae by the addition of : (*a*) an extract obtained from healthy guineapig liver by a fat-solvent [" Petroläther "], (*b*) cod-liver oil, (*c*) oleic acid, and (*d*) " vitamin F." These effects were demonstrated by mixing each substance with a virulent suspension which, after a suitable interval, was used for the intraperitoneal or intranasal inoculation of mice. Suspensions treated with appropriate doses of each of these substances were much less lethal than similar quantities of untreated suspensions. Some examples are :—Among 10 mice inoculated intraperitoneally with 1·0 cc. of a 1–40 mouse lung suspension to which 10 mgm. of liver extract had been added 90 minutes previously, there were 4 deaths 2–14 days after inoculation, and among 20 mice inoculated with the same dose of untreated suspension there were 10 deaths within 24 hours and 8 deaths after 2–14 days. The liver extract was believed to be a fatty acid. In a similar experiment in which the suspension was treated with cod liver oil 45 minutes before the inoculation of 20 mice there were 4 deaths within 24 hours and 5 within 2–14 days ; all the 20 control mice died within 12 hours. These experiments show that the substances had a pronounced antitoxic effect.

Suspensions treated with the liver extract were introduced by the nasal route into 30 mice, of which 7 died after 5–7 days, and one other at a later stage, whereas all of 13 control mice died after 3–4 days. In this case the infectivity of the suspension must have been greatly reduced by the liver extract.

When suspensions treated with oleic acid were used for nasal and intraperitoneal inoculation, similar effects were observed.

The addition of " vitamin F," an unsaturated fatty acid, to suspensions had a striking effect ; none of 20 mice inoculated by the nasal route with suspensions containing 2–10 mgm. per cc. of the vitamin died, but all the 10 control mice died after 3–4 days.

When mice already infected by the nasal route were treated by considerable doses of oleic acid administered by inhalation, no significant therapeutic effect was observed, so that no claim is made for the efficacy of the drug *in vivo* despite its pronounced action *in vitro*. *John W. D. Megaw.*

SNYDER, J. C. & WHEELER, C. M. **The Experimental Infection of the Human Body Louse,** *Pediculus humanus corporis*, **with Murine and Epidemic Louse-Borne Typhus Strains.** *J. Exper. Med.* 1945, July 1, v. 82, No. 1, 1–19, 3 pls. [23 refs.]

The authors give a detailed description of methods by which they have infected lice with typhus rickettsiae, and have maintained the lice after infection, by using rabbits for both purposes; these methods obviate the necessity for intrarectal inoculation of the lice and the use of human beings for maintaining the infected lice.

Groups of lice from colonies found free from rickettsiae were infected in two ways: (*a*) by feeding them on a "bleb" produced by injecting a small quantity of infected material into the skin of the abdomen or ear of the rabbit, and (*b*) by feeding them on rabbits which had received, intravenously, large doses of infected material, usually yolk-sac suspensions. The lice were maintained by feeding them on the skin of a rabbit after shaving it without the use of soap and then applying a little human saliva, preferably from a non-smoker.

Eight sets of experiments are described; three of these were by the "bleb" technique, four by the intravenous technique, and one by both combined.

Various materials from infected gerbils or yolk sacs, mixed with human serum, were used for the "bleb" technique: 93 lice were used and the calculated rate of survival of the lice for 10 days after feeding was 48 per cent. The routine test for infection was by examining gut smears for rickettsiae; in each set of experiments the finding was confirmed by animal inoculation. No infection was found during the first two days, but the lice examined after 3 and up to 10 days had an infection rate of 76 per cent.

For the intravenous tests, 1,174 lice were used; the calculated survival rate was 23 per cent. after 7 days. Among the lice fed on rabbits less than 15 minutes after inoculation the infection rate was 98 per cent.; examples of the infection rates among lice fed at longer intervals are:—After $\frac{1}{4}$–$\frac{1}{2}$ hour, 93 per cent.; after $8\frac{1}{2}$ hours, 68 per cent.; after 45 hours 33 per cent.; and even after 93 hours 5 per cent. No infection was found after 117 hours.

Control lice fed on healthy rabbits never showed rickettsiae; their 7-day survival rate was 95 per cent.

In a separate experiment, 2,000 lice were fed on an infected rabbit within one hour by a single worker who also continued to feed all the surviving lice twice daily for seven days. The faeces of the lice were infective for gerbils and, in a random lot of 20 lice, rickettsiae were found in 18. The worker who handled this lot of lice did not become infected; he used rubber gloves and a mask; two other workers who took no special precautions while handling infected lice had mild attacks.

It is suggested that if proper precautions are taken lice may be used for the isolation of rickettsiae from patients.

Epidemic strains of rickettsiae were used in all the above experiments except one in which seven lice were fed on a "bleb" infected with murine rickettsiae of the Wilmington strain obtained from tunica washings from an infected gerbil. Five of the lice fed on the murine strain were injected into a guineapig, which reacted with fever lasting three days and with scrotal swelling; smears from the only louse tested in this way contained no definite rickettsiae.

The original paper, illustrated by five clear photographs, must be consulted for details of the technique employed. *John W. D. Megaw.*

LINSKIS, D. Y. [**A Case of Post-Transfusion Typhus.**] *Sovetskaia Meditsina.* Moscow. 1944, Nos. 10/11, 19. [In Russian.]

The recipient was a woman who, 10 days after operation for mastoiditis, received a transfusion of 200 cc. citrated blood. Exactly 8½ days later she became febrile, and 5 days afterwards developed a rash, and the diagnosis of typhus was made. The donor of this blood was a woman who, on the third day after the blood had been taken, developed symptoms which later led to the diagnosis of typhus. This proves that the rickettsiae of typhus are present in the blood in the later stage of the incubation period. *H. W. Swann.*

ALCANTARA, A. G. Formas clinicas del tifus exantemático. [**Clinical Forms of Exanthematic Typhus.**] *Rev. Sanidad e Hig. Pública.* 1945, Jan., v. 19, No. 1, 50–66, 1 graph. [25 refs.]

The author saw about 100 cases of louse-borne typhus in hospital and private practice in Murcia between March 1941 and April 1944.

The rash never extended to the palms of the hands and it was seen on the face in only one case. The author found that the rash could often be detected at an earlier stage than usual by applying a dry cup for 6–8 minutes to the skin of the abdomen near the umbilicus. The rash was always macular, never papular; when petechiae occurred they were situated in the centre of the macules.

The classification of the cases according to the main features was—cerebral (44); respiratory (9); digestive (7); mixed types (26); and typhus without rash (14).

The respiratory cases were often wrongly diagnosed as influenzal pneumonia till the appearance of the rash indicated the real nature of the illness. The condition was regarded as a true typhus pneumonia caused by the rickettsiae; it did not yield to sulphonamides.

The digestive cases usually had diarrhoea with tenesmus and colicky pains in the umbilical region, they were usually rather mild and the termination was by slow lysis.

Among the cases without rash 11 were mild, including three inapparent attacks in infants, and three were exceptionally severe.

The author accepts the view that attacks among children, which are often exceptionally mild and likely to be missed, are of great importance in maintaining the infection in a community, especially as lice infected from these patients tend to survive for long periods and so remain capable of transmitting infection far longer than severely infected lice. *John W. D. Megaw.*

DE MAGALHÃES, O. & ROCHA, A. Tifo exantemático neotrópico no Brasil. Ensaios terapêuticos com a Penicilina. [**Typhus in Brazil: Therapeutic trial of Penicillin.**] *Mem. Inst. Oswaldo Cruz.* 1944, Aug., v. 41, No. 1, 59–64. English summary.

"The authors described the experiments with Penicillin Merck-Sodium Salt n. 135 in order to verify the curative action on the experimental disease of the guineapig.

"1. They found that small doses of 1,600 units, Oxford, when inoculated during the first 24 or 48 hours of the appearance of the feverish reaction are impotent to modify the course of the disease, whether in the thermic curve, or the testicular or splenomegalic manifestations.

"2. Penicillin, when used in larger doses, from 25,000 to 30,000 Oxford units, in 24 hours through peritoneal cavity is capable of normalising the temperature, modifying to a certain degree the virulence of the virus in the guineapig.

"3. In large doses, even when injected later, it causes the temperature to fall, in all animals, attenuating the visceral manifestations.

"4. Direct contact of Penicillin with the virus, during many hours, causes it to lose its virulence.

"They conclude that it is possible perhaps with stronger doses of the drug, given intravenously in man, to better the morbid evolution of the serious forms of Neotropic Exanthematic Typhus in Brazil, not forgetting the death-rate of the patients is from 88 to 100 per cent."

YARYGIN, N. E. & NAGIBINA, N. I. [**The Treatment of Typhus by Buxation.**] *Sovetskaia Meditsina.* 1944, No. 10/11, 17–19. [In Russian.]

"Buxation" [a Russian term] is a form of irritation of the central nervous system, and consists in repeated withdrawal and reinjection of cerebrospinal fluid through a cerebrospinal [suboccipital ?] puncture; a 10 cc. syringe is used and 10 cc. of cerebrospinal fluid is alternately withdrawn and injected 10 to 15 times, the last syringeful not being reinjected. The method was used in cases of typhus of 5–8 days' duration. On the day preceding the "buxation," the patient was given 10 doses of 0·6 gm. of sodium salicylate [orally ?] and this was repeated on the day following the "buxation." This treatment was given to 145 patients, while another 20 patients received four doses of 0·5 gm. of sulphapyridine instead of sodium salicylate. The results are presented in four groups.

I. In 33 cases all symptoms disappeared immediately after "buxation," the temperature fell by crisis, and the rash faded within two days; there was no relapse.

II. In 57 cases there was a more gradual disappearance of symptoms in about three days, and no relapse occurred.

III. In 18 cases symptoms disappeared as in Group II, but some of the symptoms reappeared, a very atypical form of typhus resulting.

IV. In 37 cases the treatment had no apparent effect.

The fatality rate in the 145 cases treated with sodium salicylate was 3·45 per cent., which is half the rate observed in cases treated symptomatically. Complications were present in 8·9 per cent. only. The other 20 cases were regarded as too few to enable an estimation of the effect of the treatment to be made. [The two queries in brackets were inserted to indicate that the original description was not quite precise.] *H. W. Swann.*

ALISOV, P. A. & KOSSOVAJA, E. M. [**Vaccine Therapy of Typhus.**] *Sovetskaia Meditsina.* Moscow. 1944, Nos. 10/11, 15–17. [In Russian.]

Vaccine treatment was given in severe cases only, in most of which complications were present, and was not continued beyond the 8th–9th day of disease. The vaccine was diluted to one-fifth of the original strength, and was injected intradermally. The first dose was 0·2 cc. divided and injected in two places. Doses were increased daily for the whole course of five days, on the last of which 1·0 cc. was divided and injected in 10 different sites. In the whole course 3·0 cc. of diluted vaccine were used. There were some local, but no general, reactions; in most cases a favourable effect was observed in that the fever began to subside and the rash did not become petechial. The average duration in patients treated with vaccine was 11·2 days, in controls 14·2. The mortality rate was only one-third of that in the series of patients treated in the ordinary way. *H. W. Swann.*

SILVA, R. & CASTAÑEDA, M. R. **Simultaneous Infection in Laboratory Animals with Murine and Classic Typhus. II. Recovery of Strains.** *Proc. Soc. Exper. Biol. & Med.* 1945, May, v. 59, No. 1, 84–6.

The authors have already produced pneumonia in mice and rats by intranasal inoculation of a mixture of murine and classic rickettsiae and have satisfied themselves that both strains of infection persisted through several passages [see this *Bulletin*, 1945, v. 42, 457]. They have now made a series of transfers through guineapigs by intraperitoneal inoculation, starting with mouse-lung suspensions obtained from animals of the 10th lung passage of the mixed infection. Through male guineapigs 18 successive passages were made; all but two of the animals had scrotal reactions; all gave strongly positive complement-fixation reactions, the titres were considerably higher when murine antigen was used than when classic antigen was used. Tunica material from the guineapig of the 10th passage was inoculated into a female guineapig which reacted feebly, but after a few passages through female guineapigs (in which brain material was used) there was a temporary increase in virulence. On transferring the infection again to male guineapigs the strain was found to cause scrotal swelling and to give a much stronger complement-fixation reaction with murine than with classic antigen.

Another series of transfers was made through female guineapigs, starting with the same mouse-lung material as was used in the other series; in the early passages the complement-fixation responses were feeble or negative, but from the 4th passage onwards reactions with classic antigen became strongly positive. Brain material from the female guineapigs, when transferred to males, caused no scrotal reactions, but after several further transfers through male guineapigs some sera gave positive complement fixation reactions with classic antigen, but none reacted with murine antigen.

It was concluded that transfers through male guineapigs favoured the isolation of murine rickettsiae and transfers through females favoured the preservation of the classic rickettsiae. *John W. D. Megaw.*

DAMON, S. R. & JOHNSON, Mary B. **The Serologic Diagnosis of Endemic Typhus. II. A Comparison of Water-Bath and Icebox Fixation in the Complement Fixation Test.** *J. Lab. & Clin. Med.* 1945, May, v. 30, No. 5, 415–19. [10 refs.]

Using various antigens, including a number of commercial epidemic-typhus vaccines, the authors have carried out a series of complement-fixation tests on the sera of rats infected with endemic typhus. They followed the technique described by BENGTSON [this *Bulletin*, 1944, v. 41, 738], but found that by keeping the mixtures of serum, antigen and complement in the icebox overnight, much higher titres were obtained than when the mixtures were incubated for one hour at 37°C. Many human sera were also tested, with similar results, and in some cases weak positives were obtained with the icebox technique when the results with the other method were negative; in these cases later tests yielded high-titre reactions with both methods, but the titres were always higher with the modified technique.

The authors consider that the method is likely to be of special value in rat surveys of localities in which endemic typhus is suspected.

John W. D. Megaw.

ARBONA, G. **Atabrine in the Treatment of Endemic Typhus Fever.** *Bol. Asoc. Med. de Puerto Rico.* 1945, June, v. 37, No. 6, 208–10, 3 charts.

The average duration of the fever in 13 cases of endemic flea-borne typhus fever in which treatment by atabrine [mepacrine] commenced 5–19 days after

the onset, was 13·9 days. In the 13 previous cases treated in the same way, but without atabrine, the average duration was 16·2 days. The average duration of the fever after admission to hospital in the atabrine-treated cases was 4·1 days ; in the controls it was 6·2 days. The usual dosage was 0·1 gm. thrice daily.

The author states that the number of cases is too small to justify definite conclusions on the value of the drug, but in the absence of any other available treatment he considers that its use is indicated.

Reference is made to the use of atebrin in the treatment of typhus fever by VAN MEERENDONK [this *Bulletin*, 1943, v. 40, 237], but in that series of trials also there were no real controls. *John W. D. Megaw.*

BLAKE, F. G., MAXCY, K. F., SADUSK, J. F., JR., KOHLS, G. M. & BELL, E. J. **Studies on Tsutsugamushi Disease (Scrub Typhus, Mite-borne Typhus) in New Guinea and Adjacent Islands : Epidemiology, Clinical Observations, and Etiology in the Dobadura Area.** *Amer. J. Hyg.* 1945, May, v. 41, No. 3, 243–373, 32 figs. (1 map) & 3 coloured pls. [152 refs.]

This important paper runs to 130 pages, it deals with a review of the literature (32 pages), epidemiology (20 pages), clinical observations (31 pages), and aetiology (33 pages) ; there is also a bibliography containing 152 references. There are 32 black-and-white illustrations, and three plates containing good reproductions of colour photographs.

The investigation was carried out under the auspices of the U.S.A. Typhus Commission and the Preventive Medical Service of the U.S.A. Army. Independent investigations made by officers of the Australian and U.S.A. forces have been freely quoted, and as reports of most of these have already been reviewed in this *Bulletin*, the present abstract will deal chiefly with the unpublished work carried out by the authors.

In the critical review of the literature the following statements are made :— (1) The name *Rickettsia orientalis* proposed by Nagayo in 1930 has the best claim to recognition ; (2) the name tsutsugamushi has precedence over other designations of the disease ; (3) the only species of mite hitherto proved by animal experiment to be a vector is *Trombicula akamushi*, though there is much evidence to incriminate *Tr. deliensis* ; and (4) the only animals yet found naturally infected are the field vole, *Microtus montebelloi*, in Japan and an undetermined species of wild rat in the Federated Malay States.

The authors investigated the mites of the Dobadura area of New Guinea and isolated *R. orientalis* from two pools of *Tr. fletcheri*, each of which was collected from a separate bandicoot (*Echymipera cockerelli*) ; neither of the bandicoots showed any evidence of being infected though a thorough investigation was made. A few *Tr. deliensis* and *Tr. walchi* [see KOHLS *et al.* below] were found ; the former only in one restricted area which " happened " to be highly infected, the latter occurred sparingly. Among 13 other species of mites found in the area *Tr. buloloensis*, *Schöngastia blestowei*, and *S. pusilla* were abundant and widely distributed ; they were known to attack man and to cause scrub itch but " it seemed quite unlikely that they served as vectors."

The clinical features of 20 proved cases of mite-borne typhus were investigated ; there were two deaths ; one patient died suddenly and unexpectedly on the 5th day, of a haemorrhage in the pons and medulla, the other was one of the two patients who had hepatitis with jaundice. The severity was very variable, ranging from mild (two cases) to severe or very severe (nine cases). There were clinical signs of pneumonitis in 13 cases ; in seven of these the condition was severe, but it was regarded as being essentially rickettsial in origin, except in one patient from whom a pneumococcus was recovered.

The incubation period ranged from 10 to 18 days. An eschar was found in every case. The rash seldom extended to the face, feet, or hands.

Among 24 cases in which the Weil-Felix reaction was studied there were seven in which the *Proteus OXK* titre never exceeded 1–40, and two others in which it was 1–80 ; in the remaining 15 it rose at some period to 1–320 or over, though in no case examined between the 4th and 11th days was a titre higher than 1–40 observed. With *Pr. OX2* all the reactions were negative ; with *Pr. OX19*, 5 were negative ; 5 were positive 1–20 ; 7 were positive 1–40 ; 4 were positive 1–80 ; and 3 were positive 1–160, but these reactions were not in rising titre and similar reactions occurred in 5 of 13 patients suffering from other diseases ; so also 3 of these 13 had titres of 1–40 against *Pr. OXK*.

Problems in differential diagnosis arose : in addition to the 20 patients in whose cases the diagnosis was confirmed, eight patients with an initial diagnosis of the disease were studied, but five of these turned out to be " proved " cases of dengue, one was doubtfully dengue, and two others were infectious hepatitis. There was still another case in which the diagnosis remained doubtful after full investigation. In four of the cases of dengue the maximum *OXK* titre was 1–40 ; the rash in five of the cases of dengue and in one case of infectious hepatitis was not " clearly distinguishable from that of mite-borne typhus." [The evidence on which the diagnosis of dengue was regarded as proved is not stated.]

In 16 cases attempts were made to recover *R. orientalis* from the blood of patients by intraperitoneal inoculation of white mice ; in four of these dengue was diagnosed ; in two others the diagnosis was infectious hepatitis, and in another the diagnosis remained uncertain ; from the remaining nine patients the organism was isolated and thoroughly studied by repeated passage through mice, which usually died between the 10th and 20th day after inoculation by the intraperitoneal route. Suspensions of blood or brain tissue of the mice, diluted 1–30 in the later passages, were always infective ; they were taken on the 7th to the 14th day. Smears of peritoneal scrapings of the infected mice always showed rickettsiae. The primary inoculation of the mice was by 0·2–0·3 cc. of a 10 per cent. suspension of blood clot in normal saline.

Intraocular inoculations with infected material from mice were carried out on 54 rabbits, of which 48 reacted in the typical manner, but none of the rabbits developed the positive *OXK* agglutinins described by LEWTHWAITE as occurring in 50 per cent. of rabbits inoculated by this route. All the six strains used for these inoculations gave positive results ; these are beautifully illustrated by six photographs in colour.

The Syrian hamster was found specially suitable for passage of the rickettsiae ; after intraperitoneal inoculation all the animals died, but after subcutaneous inoculation most of them survived and so were valuable for immunity tests because of their being insusceptible to intraperitoneal inoculation.

Yolk-sac cultures were successful in all the five strains that were tested ; great variations occurred in the number of rickettsiae seen in smears, but the yolk sacs in which very few organisms were found were infective in the same extremely high dilutions as the sacs containing numerous rickettsiae.

The two strains isolated from mites were thoroughly investigated and were found to be identical with the human strains. Full details are given of all these laboratory studies by which the identity of all the strains was established ; most of the studies were carried out in the Division of Virus and Rickettsial Diseases of the Army Medical School, Washington, after the return of the Commission to the U.S.A.

[This monograph will be found invaluable by all workers on mite-borne and other typhus fevers, it deals with every aspect of tsutsugamushi disease except

modern developments in connexion with preventive measures, which, for obvious reasons, could not be discussed at the time of writing.]

John W. D. Megaw.

BLAKE, F. G., MAXCY, K. F., SADUSK, J. F., Jr., KOHLS, G. M. & BELL, E. J. *Trombicula fletcheri* **Womersley and Heaslip 1943, a Vector of Tsutsugamushi Disease (Scrub Typhus) in New Guinea.** *Science.* 1945, July 20, 61–4. [Refs. in footnotes.]

This is a preliminary notice of the work reviewed above.

KOHLS, G. M., ARMBRUST, C. A., IRONS, E. N. & PHILIP, C. B. **Studies on Tsutsugamushi Disease (Scrub Typhus, Mite-borne Typhus) in New Guinea and Adjacent Islands : Further Observations on Epidemiology and Etiology.** *Amer. J. Hyg.* 1945, May, v. 41, No. 3, 374–96. [14 refs.]

This report describes an investigation carried out in continuation of the studies dealt with in the preceding abstract. The area investigated was New Guinea and the adjoining Islands. The types of locality found infected were :—(1) open kunai-grass land, (2) abandoned banana and coconut gardens with undergrowth of grass and shrubs, (3) sparse coarse growths of native vegetation, and, possibly, (4) areas on the edges of virgin forests.

In one area in Dutch New Guinea a single regiment had 308 admissions, including the Officer Commanding and 30 other officers. In two islands off Dutch New Guinea there were 1,056 cases within 2½ months, and one-quarter to one-third of the personnel of three battalions were attacked. The case-fatality rate varied from less than 1 per cent. to 27·5 per cent.

In the Dobadura area more than 30 species of mites were found ; 65–72 per cent. of all the mites found on rats and bandicoots in this area were *Trombicula fletcheri*, which were very abundant on the animals in grassy areas, but fewer in those found in jungle and well-cleared camp sites.

Details are given of the mites found and of their vertebrate hosts, which included birds. A few ticks were found in one affected area ; a single *Amblyomma* (species not identified) was found on each of three men. Preliminary attempts to transmit infection by *Ornithodorus moubata* failed.

One strain of *Rickettsia orientalis* was recovered from a pool of *Tr. fletcheri* taken from a rat (*Rattus concolor browni*) and three other strains were recovered from *Tr. walchi* from rats of the same species. Altogether 56 pools, comprising 17,000 mites, were tested. It is strongly suggested that *Tr. walchi* may be the same as *Tr. deliensis* Walch.

Attempts were made to infect eight locally captured bandicoots by intraperitoneal inoculation, but without success. Among four other bandicoots from a non-infected area (Brisbane), inoculated in the same way, three gave no reaction, but peritoneal washings from the 4th animal were infective to mice, though blood and brain substance of this animal was not infective. The authors comment that " this is an anomalous result without adequate explanation," and they regard bandicoots as being unlikely to be natural reservoirs of infection.

Among 36 pools of wild rats of various species, comprising 102 rats, *R. orientalis* was recovered from two pools made up of seven and four rats respectively, all belonging to the species *R. concolor browni*. Some of the rats in each of these two pools were infested by *Tr. fletcheri*. The rickettsiae were identical with those recovered from patients and mites as described in the preceding paper.

Altogether 30 strains of *R. orientalis* were recovered from patients, 15 from blood taken on the 3rd to the 10th day of the illness, and 15 from heart blood obtained at autopsies.

Infection in guineapigs was non-orchitic and it tended to die out after a few passages. In white rats the infection was inapparent, but it persisted for long periods in the brain, in one case up to 98 days at least ; it caused no rise above 1–40 in the Weil-Felix titre in the rats.

Unsuccessful attempts were made to induce larval mites captured on animals to bite other animals, so that mites attached to rats or domestic pets are not regarded as being " particularly hazardous " ; and as rats often persist in large numbers in places from which the disease has disappeared, control measures should probably be concentrated on the mites. Repellents, such as a 5 per cent. emulsion of dimethyl phthalate in 2 per cent. soap solution, have been found effective. *John W. D. Megaw.*

MACHELLA, T. E. & FORRESTER, J. S. **Mite or Scrub Typhus. A Clinical and Laboratory Study of 64 Cases.** *Amer. J. Med. Sci.* 1945, July, v. 210, No. 1, 38–61, 4 figs. & 12 charts.

The authors give an exceptionally clear and orderly description of the clinical features observed in 64 cases of mite-borne typhus during the last three months of 1943 and the first month of 1944. The patients were 40 Chinese and 24 American soldiers, all of whom had been in the jungle during the weeks immediately preceding the onset.

Only one of the patients died and no mention is made of any exceptional incidence of previous illness or severe hardship, so that the clinical picture was not likely to have been seriously affected by these disturbing factors.

The authors state that " in this paper the disease will be referred to as mite typhus, the name ' scrub typhus ' being reserved for those cases of typhus-like disease in which the vector is not reasonably clear." [The unhappy name scrub typhus is now so firmly established by usage as a synonym of mite-borne typhus that this special application of the term is not likely to be adopted.]

The average duration of the fever was 18·3 days in Chinese, and 16 days in American, patients ; of the onset symptoms, cough and epistaxis were more frequent in the former and photophobia in the latter group, otherwise no great difference was observed between the two groups. All the American patients had been inoculated against louse-borne typhus at unspecified dates, but neither the severity of the attacks nor the Weil-Felix titres seemed to have been influenced by the vaccine.

The figures shown below in parentheses refer to percentage incidence.

The primary lesion (67) was usually on the upper part of the trunk, mostly in the axillae or on the left costal margin ; it appeared first as a red macule which in a few days became a papule, soon being replaced by an ulcer filled by a black eschar. In all but four cases the local lesion was accompanied by regional lymphadenitis. The onset was sudden, though sometimes the temperature rose gradually, the chief features being fever (90), anorexia (88), headache (76), chill (68), conjunctival injection (53), cough (40), and backache (25). Apathy was pronounced but the eyes were bright and alert as in louse-borne typhus.

The types of temperature curve were " typhoidal, relapsing, remittent, or intermittent." One patient was regarded as having had a true relapse, his temperature had become almost normal on the 12th day, but on the 13th day it began to rise again, though the fever continued to be remittent, and there was a recurrence of splenomegaly. The total duration of fever was 24 days, and the Weil-Felix reaction did not become positive till the 21st day of the disease.

The rash (51·5) was usually maculo-papular but sometimes it was purely macular ; the palms and soles were never involved. The spleen was palpable in 88 per cent. of the Chinese patients, and occasionally it was also tender. Generalized lymphadenopathy (93·7) appeared during the first week. Distension of the retinal veins was seen in 30 Chinese and 14 American patients about the 7th day : it was followed by oedema of the optic disk in 18 Chinese patients, of whom four developed retinal haemorrhages. Among the 40 Chinese patients X-ray examination showed the presence of bronchitis in 9, of bronchopneumonia in 9, and of both these conditions in 4. The authors could not be certain whether the pulmonary involvements were complications or part of the disease. [Probably the latter, sulphadiazine was given to some of these patients, without effect.] Acute mania occurred in one patient on the 7th day. Muscular twitchings were observed in 4 cases, generalized convulsions in 2, and meningismus in one patient whose spinal fluid contained 60 leucocytes per cmm. and a trace of globulin ; in this and two other cases the fluid was at high pressure.

Among 12 patients examined by electrocardiography, one had indications suggesting myocardial damage. The Weil-Felix reaction (with *Proteus OXK*) did not become positive till after the end of the 2nd week in 29·7 per cent. of the Chinese patients ; in two cases not till the end of the 6th week. Titres exceeding 1–200 were observed in 15 of 25 patients who had primary ulcers, and in only one of 15 who had no ulcer.

The leucocyte picture was very variable ; a moderate degree of leucopenia and a gradual increase in the percentage of lymphocytes were frequent findings. Mild or moderate azotaemia occurred during the 2nd or 3rd week in 13 of the Chinese, and three of the American, patients. The patient who died had appeared to be doing well except for a rapid rise in his blood urea nitrogen, which reached 108 mgm. per 100 cc. on the 16th day when he suddenly had generalized convulsions with coma, soon followed by death in a state of shock.

In 19 Chinese and six American patients there was some degree of albuminuria with granular or hyaline casts ; in five cases a few red blood cells were found in the urine.

Treatment was mainly symptomatic and supportive, and general measures included adequate intake of fluid and calories, by mouth whenever possible. Sulphadiazine was given to 10 patients without obvious benefit. Convalescent blood, given in a dose of 500 cc. to two patients on the 5th day, did not appear to influence the course of the disease. Oxygen was given for cyanosis, and blood transfusion for the shock state. *John W. D. Megaw.*

FISCHBACH, W. M. & HOWELL, D. E. **Observations on Tsutsugamushi Disease.** *U.S. Nav. Med. Bull.* 1945, Sept., v. 45, No. 3, 423–8, 2 graphs.

BRAZZAVILLE [A. E. F.] : RAPPORT SUR LE FONCTIONNEMENT TECHNIQUE DE L'INSTITUT PASTEUR EN 1943 [CECCALDI, J., Director]. 87–8. La réaction de Weil-Félix chez les chiens de Brazzaville. [**The Weil-Felix Reaction in Dogs in Brazzaville.**]

The sera of forty-six dogs, some of which belonged to Europeans who had recently had fever with a rash of the typhus type, were tested against *Proteus OX19, OX2, OXK* and *OXL*. Positive results were found in seven : 5 with *OX19* at 1–50 ; 4 with *OX2* at 1–50 ; 3 with *OXK* at 1–100 ; 1 with *OXL* at 1–100.

It is concluded that infection of the *boutonneuse* type can stimulate in dogs agglutinins for the various strains of *Proteus*. *Charles Wilcocks.*

MILNE, R. I. **A Note on the Incidence of Sandfly Fever in the Naval Camp at Haifa.** *J. Roy. Nav. Med. Serv.* 1945, July, v. 31, No. 3, 179–80, 1 graph.

FITZPATRICK, Florence K. **Penicillin in Experimental Spotted Fever.** *Science.* 1945, July 27, 96–7.

Each of six guineapigs, whose average weight was about 500 gm., received 500 units of penicillin intramuscularly every 4 hours for 4 days ; the treatment began 48 hours after the onset of fever resulting from an intraperitoneal inoculation with a virulent strain of Rocky Mountain spotted fever rickettsiae. All the guineapigs died.

Six guineapigs inoculated in the same way received 1·0 cc. of " spotted fever rabbit immune globulin " 48 hours after the onset of the fever, and all recovered.

Among 16 control guineapigs inoculated in the same way and receiving no treatment there were eight recoveries.

The penicillin-treated guineapigs showed all the typical reactions that follow inoculation with a virulent strain of the spotted fever rickettsiae, and the higher mortality among the treated animals was probably due in part to the toxicity of the drug.

Estimates of the penicillin content of the plasma of the treated animals showed that the treatment would have been adequate if it had been of value ; the dosage corresponded to a total of 2 million units for a person weighing 160 pounds. *John W. D. Megaw.*

BARTONELLOSIS.

UBATUBA, F. & VIEIRA, G. Estudos sôbre a bartonelose. I. A bartonelose dos ratos esplenectomizados e a Penicilina. [**Studies on Bartonellosis. I. The Effect of Penicillin on the Bartonellosis of Splenectomized Rats.**] *Mem. Inst. Oswaldo Cruz.* 1944, Aug., v. 41, No. 1, 21–44, 8 figs. [55 refs.] English summary.

The experiments, the upshot of which is here related, were carefully carried out, and though the result was negative it is none the less important. After reviewing published work on the anaemia of bartonellosis in rats, the author proceeds to show that 95 per cent. of the white rats at the Institute develop bartonellosis after splenectomy and that the fall in the haemoglobin coincides with the appearance of the *Bartonella* in the peripheral blood. This drop in the haemoglobin is more rapid than the reduction in red corpuscles, and when regeneration takes place the haemoglobin recovery is quicker than that of the corpuscles, but death results in about half the animals, and haemoglobinuria is a bad prognostic sign. Attempts to ward off the fatal issue by the use of penicillin were ineffectual. *H. Harold Scott.*

DENGUE.

KAPLAN, A. & LINDGREN, A. **Neurologic Complications following Dengue.** *U.S. Nav. Med. Bull.* 1945, Sept., v. 45, No. 3, 506–10.

In a hospital in the Central Pacific, 1,488 cases of dengue were seen within a period of 40 days. Soon after recovery—usually about two weeks, but in

one case only three days, and in another a month—13 of the patients had unilateral attacks of neuritis. The nerves affected were :—The facial (Bell's palsy) in two cases, the palatine, long thoracic, and ulnar in two cases each, the peroneal nerve in four cases, and the sciatic nerve in one case.

Most of the patients were evacuated and could not be followed up, but one of the patients who had Bell's palsy had recovered six weeks later, in one case of palatine-nerve palsy there was great improvement within three months, and in one case of ulnar-nerve palsy improvement was pronounced within two weeks. On the other hand, no improvement had occurred in either of the cases of long-thoracic-nerve palsy after two and three months respectively, and in the case of peroneal-nerve palsy that could be followed up there was no improvement five weeks after the onset.

The attack of sciatic neuritis began three days after recovery from dengue ; no improvement had occurred within three weeks.

All the patients were treated with thiamin, though there was no reason to suspect vitamin deficiency ; all of them had received tetanus toxoid four to twelve months previously. It was thought likely that other cases might have occurred. *John W. D. Megaw.*

BACILLARY DYSENTERY.

ROSE, T. F. **The Clinical Differentiation of Appendicitis and the Dysenteries.** *Med. J. Australia.* 1945, July 21, v. 2, No. 3, 72–7.

The author has been impressed by the number of cases of acute appendicitis in the tropics in which diarrhoea is prominent and may resemble that of early bacillary dysentery.

He has encountered cases of bacillary dysentery which simulate appendicitis. He, further, has had cases of amoebic dysentery which simulated appendicitis ; and on one occasion, he found an obstructive appendicitis to be a sequel of a *Schistosoma japonicum* infection of the gut wall.

Detailed reports are given of 14 cases illustrating his points, and he emphasizes the importance of carefully taken histories and [far more important] of careful physical and pathological examinations in order that errors in diagnosis may be avoided. *A. R. D. Adams.*

MORTON, H. E. & ENGLEY, F. B., Jr. **Dysentery Bacteriophage. Review of the Literature on its Prophylactic and Therapeutic Uses in Man and in Experimental Infections in Animals.** *J. Amer. Med. Ass.* 1945, Mar. 10, v. 127, No. 10, 584–91. [46 refs.]

This is an excellent critical review compiled with the object of evaluating existing evidence relating to the prophylactic and therapeutic use of dysentery bacteriophage. It is itself a summary which should be read in the original. In adequate trials there must be bacteriological confirmation of all cases, and the phage used must be shown to be potent *in vitro* against the infecting strain. Since dysentery mortality is low, the best criterion of effectiveness is the time taken to clear the organism from the faeces. This test may be vitiated by the presence in the faeces of natural or administered bacteriophage which may inhibit the growth of dysentery bacilli in culture, so that the formaldehyde method of KLIGLER *et al.* [*Bulletin of Hygiene*, 1943, v. 18, 950] should be used.

The authors examine nine reports which purport to show that dysentery phage is therapeutically ineffective in man. Discarding those trials in which the groups were too small to have scientific value or the control group was

inadequate or the bacteriological investigations were open to criticism, two reports are regarded as worthy of further consideration. One is a trial described by KESSEL and ROSE [this *Bulletin*, 1933, v. 30, 762] who found, in comparison with an untreated control group, no significant difference in case-mortality or length of stay in hospital of bacteriologically confirmed cases of Flexner dysentery treated by a phage shown to be potent *in vitro*. The numbers are, however, too small and the suggested clearance test would have been a better criterion. The other trial is that of BOYD and PORTNOY in North Africa [this *Bulletin*, 1944, v. 41, 569]. They found no significant difference in the percentage of their treated and untreated individuals who developed dysentery or required admission to hospital, and no difference in the duration of stools containing blood and mucus. The average stay in hospital of the treated group was 2·86 days less than the untreated group. The reviewers comment on the low titre of the phage used.

Of 19 reports which have been cited as evidence in favour of the therapeutic value of dysentery phage, few, if any, are able to withstand criticism for reasons similar to those noted above. The most extensive trial is that of MELNIK, KHASTOVITCH and NIKHINSON (*Ann. Bull. Metchnikov Inst.*, 1935, v. 1, 97) who observed a mortality of 1·4 per cent. in 282 cases treated with Shiga phage compared with that of 2·5 per cent. in 1,059 untreated controls. It was claimed that, of the treated cases, 55·3 per cent. left hospital within 4 days, compared with 18·8 per cent. of the control group. Bacteriological cure was, however, not determined, so these results may indicate only alleviation of symptoms. These workers noted that recovery might be delayed in cases in which the dysentery bacillus was associated with *Proteus* or enterococci. This may be an important factor in the evaluation of treatment.

The verdict on the therapeutic value of bacteriophage in dysentery in man must therefore be " not proven."

In estimating the prophylactic value of dysentery phage it must, of course, be established that the phage which was used was active against the strains subsequently encountered. Four reports examined are regarded by the authors as showing that phage is capable of preventing bacillary dysentery in man. The most encouraging results were obtained by ASHESHOV *et al.* [this *Bulletin*, 1931, v. 28, 863]; MELNIK, NIKHINSON and KHASTOVITCH (*Ann. Bull. Metchnikov. Inst.*, 1935, v. 1, 89); and KLIEWE and HELMREICH (*Münch. med. Woch.*, 1941, v. 88, 617). Boyd and Portnoy [above] obtained less striking results in tests in which they used phage captured from the enemy on prisoners of war in North Africa.

The effect of phage on experimental dysentery infection in animals has been studied by a number of workers and, during the past two years, reports have been published by RAKIETEN and RAKIETEN (*J. Bacteriology*, 1943, v. 45, 477); MACNEAL, BLEVINS and PACIS (*J. Bacteriology*, 1943, v. 46, 111); DUBOS, STRAUSS and PIERCE (*J. Exper. Med.*, 1943, v. 78, 161); MORTON and ENGLEY (*J. Bacteriology*, 1944, v. 47, 475); and MORTON and PEREZ-OTERO (*J. Bacteriology*, 1944, v. 47, 475). These reports show, without any doubt, that bacteriophagy can take place *in vivo* and protect developing chick embryos and white mice against fatal infections with dysentery bacilli; that phage increases in amount *in vivo*; that it is not eliminated from the blood in 24 or 48 hours; and that quite small quantities are sufficient to have a protective effect.

The authors point out that it is illogical that tests in man were made before the animal experiments, and they consider that a carefully planned prophylactic and therapeutic trial in human beings might now appropriately be made.

J. C. Cruickshank.

AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS.

BALAMUTH, W. & SANDZA, J. G. **Simple, Standardized Culture Medium for Physiological Studies on** *Entamoeba histolytica. Proc. Soc. Exper. Biol. & Med.* 1944, Oct., v. 57, No. 1, 161–3.

After various attempts to produce a simple, easily prepared medium for cultivation of *E. histolytica*, the authors have arrived at the following :—Two eggs are boiled for 15 minutes ; after cooling, the yolks are separated and crumbled in 125 cc. of 0·8 per cent. sodium chloride solution. The mixture is boiled for 10 minutes and, after evaporated water has been replaced, the infusion is filtered by suction. The filtrate is again brought to 125 cc. by addition of water and autoclaved for 20 minutes at 15 pounds pressure. After cooling, the slight precipitate is removed by simple filtration. To the filtrate is added 125 cc. of M/15 phosphate buffer (*p*H 7·5), making the total salt concentration M/30 phosphate solution in 0·4 per cent. sodium chloride. The final mixture is distributed in test tubes in 5 cc. amounts, autoclaved and stored under refrigeration. Before a tube is inoculated with amoebae, a loop of sterile rice starch is added to it. The sterile medium thus prepared is almost completely transparent. The growth of amoebae is uniformly good, the maximal growth being attained on the second to the fourth days, and active amoebae persisting for more than nine days. Though the growth was not so intense as in some other media it was particularly suitable for studies in cellular biology. Better growth could be obtained by doubling the concentration of egg yolk or by adding 0·5 per cent. of Wilson liver concentrate powder (Wilson Laboratories, Chicago). *C. M. Wenyon.*

STOLL, Alice M., WARD, P. A. & MATHIESON, D. R. **The Effect of Ultraviolet Radiation on Cysts of** *Endamoeba histolytica. Science.* 1945, May 4, 463–4.

The radiation employed was from two types of lamp emitting energy of wavelength 2,537Å. Cysts were suspended in distilled water in concentration of 30,000 per litre. After irradiation the suspensions were sedimented, and the deposit was planted into suitable media. It was found that with an exposure of ten minutes all cysts were destroyed. When heavier suspensions were exposed for shorter intervals only a portion of the cysts were destroyed. In these cases, however, the resulting culture amoebae were abnormally sluggish, suggesting a sub-lethal effect of ultra-violet radiation.

C. M. Wenyon.

DEL SEL, M. Absceso amibiano de base del pulmon derecho con pleuresia reaccional de gran cavidad, vaciada esta por vomica. [**Amoebic Abscess of the Base of the Lung.**] *Rev. Asoc. Med. Argentina.* 1945, July 30, v. 59, No. 562, 849–51, 6 figs.

WAR OFFICE. ARMY MED. DEPT. BULL. No. 51. 1945, Sept., 2–3. **Treatment of Amoebic Dysentery.**

" Treatment of amoebic dysentery should begin as soon as possible after the diagnosis has been made and the patient has been got under conditions that allow his treatment to be satisfactorily carried out. According to the stage of the disease when it comes under treatment, one or other of the three courses A, B or C, is recommended.

" *Course A* (*for acute cases with vegetative E. histolytica in the stools*).—Emetine hydrochloride grain 1 daily by hypodermic injection for 4 to 6 days is usually enough to control the acute symptoms. Without any interval, this

should be followed by the oral administration of Emetine-Bismuth-Iodide (E.B.I.) in doses of grains 3 daily for 12 consecutive days. Great care should be taken to ensure that the E.B.I. is given in suitable form. Inspection of the stools should be made regularly to see that the capsules are not being passed undissolved; it may be emphasized that E.B.I. must blacken the stools if the capsules are disintegrating properly. Along with the E.B.I. a chiniofon retention-enema, preceded by an alkaline wash-out, should be given daily. The daily enema of chiniofon (quinoxyl, yatren, quiniosulphan) should at first be 200 c.cm. of a 2½ per cent. solution, but the quantity and strength may with advantage be increased if the enema is well tolerated. The important point is that the enema should be retained by the patient for at least six hours and to ensure successful retention he should be kept at rest during this period.

"When this stage of the treatment has been completed, one 0·25 gramme tablet of stovarsol or carbarsone should be given twice daily for 12 days. If diodoquin is available it should be used in this after-treatment instead of stovarsol or carbarsone; 3 tablets, each containing grains 3·2 (0·2 gramme) should be given three times a day for 20 days. During this stage, the patient may be allowed to get up and begin his convalescence.

"Amoebic and bacillary dysentery often occur together and this complication may be suspected on clinical grounds from the presence of a particularly acute attack. In this circumstance it is of benefit to give sulphaguanidine or sulphasuxidine (succinyl sulphathiazole), 5 grammes 4-hourly, along with the emetine injections.

"*Course B* (*for patients who are passing cysts but not vegetative forms of E. histolytica and who present neither acute dysenteric symptoms nor indications of hepatitis*).—The hypodermic injections of emetine are omitted. Treatment by oral E.B.I. with retention-enemas and the follow-up course of stovarsol, carbarsone, or diodoquin, is begun at once and carried through as in course A.

"*Course C* (*for intractable cases that resist ordinary treatment*).—Cases of amoebic dysentery that resist course A will usually respond to it after a preliminary course of penicillin and sulphasuxidine has eliminated secondary pyogenic organisms which have become established in the diseased bowel-wall. Best results have been secured by giving 100,000 units of penicillin intramuscularly as an initial dose and continuing treatment with 33,000 units every 3 hours until 2 million units have been given. The sulphasuxidine is given concurrently in 5 gramme doses every 4 hours until 80 grammes have been given. If necessary, sulphadiazine, sulphathiazole, or sulphaguanidine may be used in appropriate dosage as substitutes for sulphasuxidine.

"It should be realised that there are exceptional cases which resist these lines of treatment. The best plan in this event is to allow a suitable interval and then repeat the whole course.

"*Criteria of cure.*—Amoebic dysentery is notoriously liable to relapse. Stools should be examined macroscopically at regular intervals during treatment and microscopically on six consecutive days at least 2 weeks after all treatment has been completed; this interval between the end of treatment and the beginning of the laboratory clearance tests is important. Sigmoidoscopic examination for test of cure should likewise be deferred until the same time.

"*General considerations.*—Correctly given drug-therapy should be supported by a cheerful ward-atmosphere and good nourishing food. Milk may be given from the first and followed as soon as possible by a nutritious low-residue diet of high caloric value. Vitamins in some form—multivite tablets, for example—may sometimes have to be added."

War Office. Army Med. Dept. Bull. No. 51. 1945, Sept., 3–4. **Tre at ment of Hepatic Amoebiasis.**

"Only the vegetative stages of *Entamoeba histolytica* are ever found in a liver abscess; cysts have never been discovered there. This is fortunate for treatment because the vegetative stage is far more amenable to emetine injections than the cysts. It is largely for this reason that most cases of hepatic amoebiasis, even if there is early pus formation, will respond to a relatively short course of emetine injections. It must not be forgotten, however, that the underlying intestinal condition will almost certainly need longer and perhaps repeated courses of combined treatment.

"Emetine hydrochloride grain 1 should be given daily by injection for 9–12 days. If the symptoms are not relieved and pus is suspected, the liver should be needled. A 20 c.cm. syringe with wide-bore needle 3½ to 4 inches long is the best instrument for this purpose. Unless there is some special indication, such as a point of local tenderness or a local area of oedema, the puncture is best made at the site of election—that is, the 8th to 10th interspace in the mid-axillary line. Premedication with morphine and hyoscine or some other sedative is usually advisable; local anaesthesia is employed for the needling. If pus is found on exploration, an effort is made to drain the abscess cavity with a Potain's or other closed aspirator. Several such aspirations may be necessary at intervals of a week or more. In some cases repeated aspirations fail to effect a cure and the patient continues to go downhill; use must then be made of some form of closed suction drainage or, as some surgeons prefer, open drainage with removal of one or more rib sections. It must be remembered that there may be more than one abscess. It is worth noting that after pus has been evacuated from a large abscess shrinkage of adjacent liver tissue may greatly raise the cavity. Later aspirations must then be done at a higher level; or, with open drainage, a new and higher incision may become necessary."

Silverman, D. N. & Leslie, A. **Toxic Effects of Diodoquin.** *J. Amer. Med. Ass.* 1945, Aug. 11, v. 128, No. 15, 1080–81.

Diodoquin (5, 7-diiodo-8-hydroxyquinoline), since its introduction for the treatment of amoebiasis, has been regarded as non-toxic even when taken in large doses for a long period. Many writers have noted this. The present record is, therefore, interesting and important in showing that the drug cannot always be taken with impunity. The dose originally recommended was one or two tablets, each of 0·2 gm. (3·2 grains) three times daily; but it was found that larger doses, 3–4 tablets thrice daily, gave better results and did not cause toxic effects.

The present authors put on record three cases. Two were given four tablets three times daily, the first for 19 days, the second for 8 days (with emetine injections for five and four days respectively). Both developed a furunculosis necessitating incisions and drainage. The third patient was a woman of 51 years; she received the same daily dose of 12 tablets for 23 days. She then complained of sore throat and chilliness, and a blotchy erythema appeared, involving the whole body. This cleared up in about a week. Later examination of the stools of these patients failed to show amoebae, in the last case as long as one year after treatment. *H. Harold Scott.*

Payne, A. M. M.; Coghill, N. F. **Amoebic Dysentery.** [Correspondence.] *Lancet.* 1945, Sept. 8, 319.

i. In a previous paper [this *Bulletin*, 1945, v. 42, 381] Payne reported that sulphapyridine and sulphaguanidine allayed symptoms in chronic amoebic

dysentery in India without affecting the amoebae; in this country [Britain] he has recently treated six cases of chronic amoebic dysentery and four cases of ulcerative colitis with succinyl sulphathiazole ["sulfasuxidine"] and penicillin as advocated by HARGREAVES [this *Bulletin*, 1945, v. 42, 895].

In the dysentery cases the results confirmed Hargreaves' claims and two of the ulcerative colitis cases improved greatly and rapidly but the author is not confident that they were cured; the other two cases showed only slight improvement.

Both in India and in this country the medical officer should satisfy himself that hospital orderlies give the enemata properly as they are sometimes careless and inefficient.

ii. During the last two years Coghill has treated 70 acute and chronic cases of amoebic dysentery in the Middle East with 12 daily injections of 1 grain of emetine together with sulphonamides by the mouth; the sulphonamides were usually sulphaguanidine (3·5 gm. four-hourly for 10 days—total, 220 gm.) together with sulphathiazole (0·5 gm. five times a day for 7 days). The results are not given, being still incomplete, but the author supports the opinion of Hargreaves and suggests, further, that bactericidal drugs should be given concurrently with emetine not only in chronic amoebic dysentery but in all forms of the disease. *J. F. Corson.*

D'ARCANGELO, D. Contributo allo studio della Lambliasi intestinale in Addis Abeba. Nota N. 1. [*Giardia* **Infection in Addis Ababa.**] *Boll. Soc. Ital. di Med. e Igiene Trop.* (Sez. Eritrea). 1944, v. 4, No. 3, 465–74. English summary (4 lines).

——. Contributo allo studio della Lambliasi intestinale in Addis Abeba. Nota N. 2. *Ibid.* 475–85. [27 refs.] English summary.

The author, having had under observation 71 cases of *Giardia* infection in Addis Ababa, publishes his results together with a review of a selection of the literature dealing with this subject. He finds that infection always results in a duodenitis, associated with colitis in 30–40 per cent. of the cases. In some cases, especially in children, symptoms of dysentery occur. Atebrin is regarded as a specific remedy for the infection but it is stated that this drug acts only on the vegetative forms and not on the cysts, for which it must be given per rectum. [Such a statement suggests that the author does not realize that if all vegetative forms are destroyed cysts are no longer produced. It is no more necessary to destroy cysts of *Giardia* in the intestine than cysts of *Entamoeba histolytica* or eggs of intestinal helminths in order to bring about a cure.] *C. M. Wenyon.*

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

SWYER, G. I. M. **Rat-Bite Fever due to Cat-Bite. Satisfactory Response to Penicillin after Failure of Arsenotherapy.** *Brit. Med. J.* 1945, Sept. 22, 386–7, 1 chart.

The clinical symptoms are described in detail and are fairly typical of the disease, but the most aberrant and striking feature of the case was a severe and recurrent myositis, with pain and swelling in various muscles, which rapidly subsided after treatment with neoarsphenamine, but reappeared several times until finally cured by penicillin. The patient became ill about three weeks after being bitten and was admitted to hospital a week later.

Direct blood examination during a febrile attack on the 16th day was negative, but during the next febrile period blood was inoculated into mice, which showed spirilla a week later.

Neoarsphenamine (0·45 gm.) was given intravenously, and the next day the temperature fell to normal, but 15 days later there was a relapse and a second injection (0·6 gm.) was given, followed by a third dose eight days later. The patient was then discharged, but three weeks later there was a return of symptoms and he was readmitted to hospital and given four more injections of neoarsphenamine. After about six weeks the symptoms had completely disappeared and the patient himself claimed to be feeling quite well.

Three weeks later he was readmitted a third time, five to six months after the original infection, and on this occasion was treated with penicillin, 15,000 Oxford units three-hourly by intramuscular injection for five days. The temperature was 100·8°F. on admission, rose to 104°F. the following day, but within 24 hours it had fallen to normal and remained so; within four days the patient was feeling perfectly well.

Finally the author comments on the unsatisfactory name, "rat-bite fever," which is used for two different infections, and suggests the term "spirillosis" as being less objectionable. *E. Hindle.*

THOMPSON, R. B. **A Case of Rat-bite Fever.** [Memoranda.] *Brit. Med. J.* 1945, Sept. 22, 388, 1 fig.

YAWS.

DA CUNHA, A. M., LEÃO, A. E. A., GUIMARÃES, F. N. & CARDOSO, H. T. Ensaios terapêuticos com penicilina. I. Bouba (Framboesia, Pian. Yaws). Nota prévia. [**The Treatment of Yaws with Penicillin.**] *Mem. Inst. Oswaldo Cruz.* 1944, Apr., v. 40, No. 2, 195–200, 4 pls. English summary.

"The first results of treatment of yaws by Penicillin are here reported.

"In seven patients a complete disappearance of the external lesions was obtained between the 12th and the 44th day of treatment, and serological reactions were negative on the 60th day in all cases. The observations of the patients will be continued for six months, under immunologic controls each 8 days.

"The treatment has been performed, using a mean dosage of 200 Oxford units each 4 or 6 hours. The total amount of units per subject, has varied from 9,600 to 52,000.

"No toxic phenomena due to the drug have been observed, in spite of the frequency and the high number of injections."

DA CUNHA, A. M., GUIMARÃES, F. N., LEÃO, A. E. A. & CARDOSO, H. T. Ensaios terapêuticos com penicilina. III. Bouba (Framboesia, pian, yaws). Penicilina de procedência americana, empregada em doses baixas também cura aparentemente esta enfermidade. [**The Treatment of Yaws with Penicillin.**] *Mem. Inst. Oswaldo Cruz.* 1944, Oct., v. 41, No. 2, 247–55, 5 figs. English summary.

"Five patients of yaws were treated with American penicillin (Squibb, Winthrop and Lilly), in small doses. The treatment has been performed,

using 200 Oxford units each 4 hours. The total amount of units per subject, has varied from 24,000 to 54,000.

"A complete disappearance of the external lesions was obtained between the 12th and the 25th day of treatment."

LEPROSY.

MUIR, E. **Future Programme of B.E.L.R.A. A Progressive Plan for the Control of Leprosy in the British Colonies.** *Leprosy Review.* 1945, Aug., v. 16, No. 1, 14–21.

During the first twenty-one years since the foundation of the British Empire Leprosy Relief Association (a short history of whose work has recently been published in pamphlet form by B.E.L.R.A.) the Association has been mainly engaged in the pioneer work of demonstrating the value of the improved treatment of leprosy through the injection of soluble preparations of chaulmoogra oils, and in epidemiological studies to determine the simplest methods of finding and treating the early stages of the disease, which are alone amenable in large numbers to that treatment. The history above referred to demonstrates the success of these modern methods of control in Nigeria, British Guiana, Ceylon, India, etc., and opens the way to their more general adoption by the Governments of the many leprosy-infected areas of the British Empire. A new situation has arisen since the Colonial Development and Welfare Scheme recognized the value of the anti-leprosy work already accomplished, and provided grants towards extending the work. The most noteworthy of these is the sanction of an expenditure during the next five years of £258,000 to cover the capital and recurrent costs of a new leprosy department of the Nigerian Government, to take over the doctors, nurses and lay workers of the very successful leprosy agricultural colonies, which have been largely organized and financed hitherto by B.E.L.R.A. and various missionary bodies. This will free £3,500 a year and thus enable the Association to extend its work in other Colonies, especially those of severely infected African tropical countries, and also to extend research work with a view to carrying out prolonged and carefully controlled tests of sulphonamide and other promising drugs with a view to establishing a more effective treatment of the difficult infective lepromatous types of leprosy, which, if successful, will hasten very greatly the reduction, and eventual eradication, of the disease from a great part of our empire.

After pointing out the great opportunities thus opened out of still more effective work for B.E.L.R.A. as soon as doctors and others become available for employment under the Association, to finance whom a special appeal is being made this year, Dr. Muir gives in this paper a brief survey of the most urgent requirements of the following British Colonies and Territories. Nigeria presents the most serious Empire problem outside India, on which B.E.L.R.A. has especially concentrated its efforts in recent years. A number of additional medical and lay staff will be required to extend the work; they can best be recruited and trained by B.E.L.R.A. The Equatorial province of the Anglo-Egyptian Sudan presents another large problem, where work by the Government and by the C.M.S. is already in progress, but requires material extension.

In East Africa much pioneer work has been done by medical missionaries with B.E.L.R.A. help, but only the surface of the problem has been touched ; plans for greatly extending the work only await the necessary staff and funds. In West Africa very little has yet been done in the Gold Coast, Sierra Leone and the Gambia. In the West Indies the Colonial Development Fund is now giving assistance, but more workers are required, and the same remark applies to several smaller infected areas. There is thus ample scope for further work by B.E.L.R.A. *L. Rogers.*

SHAMA RAO, A. **Leprosy Surveys in Hyderabad, Deccan.** *Leprosy in India.* 1945, Apr., v. 17, No. 2, 64–8, 1 map.

This is the third and concluding report of a leprosy survey of the Hyderabad State in the Deccan, and the results of the whole work are illustrated by a map showing the incidence of the disease in each of the 15 districts. From this and the tables of data it appears that the east central districts have the highest rates (0·5 to 1·0 per cent.) and western districts the lowest (0·1 to 0·3 per cent.). The north and south-eastern districts show the intermediate rates of 0·31 to 0·49 per cent. No explanation is given of these differences. The rates found by complete surveys of the population of the villages within a five mile radius of leprosy clinics at hospitals or dispensaries, were from 10 to 40 times higher than the figures of the censuses made in these areas in 1931. This is due to the number of early cases found by the survey parties. Males numbered three times as many as the females found by the surveys, but owing to the purdah system and the want of a female doctor in the survey party, this ratio is not a true picture of the actual conditions. The percentage of lepromatous cases showed the high figures of 31 to 43 per cent. of the total cases. The proportion of children below the age of fifteen was above 10 per cent. in only one of the three districts now reported on. *L. Rogers.*

LEPROSY IN INDIA. 1945, July, v. 17, No. 3, 88–100, 1 graph. **Annual Report of the Indian Council of the B.E.L.R.A. for the Year 1944.**

Much of the work referred to in this report has already been published and dealt with in previous abstracts, but the following additional points are worthy of mention. The work has been maintained in spite of war difficulties. Many army medical men have been trained in the diagnosis and treatment of leprosy and during the year 177 patients have been referred to the Leprosy Department for diagnosis ; in 126 of these the presence of leprosy was confirmed. Research work included long-term study of selected cases to correlate clinical, histological and immunological findings, and intensive leprosy surveys in selected areas of India to study racial, climatic and other factors influencing the incidence of the disease. It was thus found that a comparatively high lepromatous rate may be accompanied by low gross incidence, and a low child rate, in areas where some sort of isolation is practised, and *vice versa.* This indicates a reduction in new cases where isolation is attempted and where the people are apprehensive regarding the spread of leprosy. Teaching, publishing and propaganda work have been continued. The financial position of the association continues to be satisfactory. Immunological studies have been extended to trials of the reactions produced by non-lepromatous acid-fast bacilli ; but such preparations have not yet proved of value for the lepromin test, which would be simplified if they could be used, because they are easily cultivated. Sulphapyridine has not proved to be of value in the treatment of rat leprosy. *L. Rogers.*

DE SOUZA-ARAUJO, H. C. Infecção de ratos brancos com suco ganglionar de leproso, seguida do isolamento dum bacilo acido-alcool resistente de orgãos do murideo, em meio de Loewenstein. Nota prévia. [**Infection of White Rats with Gland-Juice from a Leper. Isolation of an Acid-fast Bacillus from the Tissues of the Rat.**] *Mem. Inst. Oswaldo Cruz.* 1941, v. 36, No. 3, 379–85, 15 figs. on 4 pls.

A man of 26 years presenting extensive leprous lesions on face, ears, trunk and limbs, and enlarged cervical and inguinal glands. Pus from one of the inguinal glands was found to be rich in Hansen's bacilli and a white rat was inoculated with it, subcutaneously in the axilla, as recommended by Marchoux. Five months later the rat was killed ; the axillary and inguinal glands were enlarged and contained bacilli morphologically resembling Hansen's organism. The patient's glands were again punctured and one cc. of pus obtained, emulsified in 3 cc. saline and injected, 1 cc. into each of three white rats. Fifteen months later one rat died and showed bacilliferous lesions in the spleen. Two months after this the second rat died and its glands and viscera were rich in acid-fast bacilli, and tubes of Löwenstein's medium were inoculated and a culture obtained. Eighteen months and one week after the inoculation the third rat died, with areas of alopecia and marked glandular enlargement with many bacteria. Sections of the organs did not reveal any of these bacteria. More tubes of the medium were sown with the gland-juice and fifteen days later there was a yellow-coloured growth consisting of acid- and alcohol-fast organisms, singly and in masses [see the following abstract].

H. Harold Scott.

DE SOUZA-ARAUJO, H. C. Cultura cromogênica dum bacilo ácido-álcool resistente isolado de pus de lesão fechada de lepra humana. [**Chromogenic Growth of an Acid- and Alcohol-fast Organism obtained from a Human Leprosy Lesion.**] *Mem. Inst. Oswaldo Cruz.* 1942, v. 37, No. 1, 29–34, 2 pls. (1 coloured). English summary p. 32.

A boy of 7 years, both of whose parents were lepers, showed numerous leprous manifestations in various parts of his body. One of these which fluctuated was opened with a galvano-cautery and pus was evacuated in which Hansen's bacilli were present in large numbers, and no other organism seen. Inoculation was made with this in tubes of Löwenstein's medium and also into guineapigs and white rats. In ten weeks three of these tubes (and also two of another lot which had been sown less than 3 weeks before) contained an abundant growth consisting of masses of acid- and alcohol-fast organisms, and subcultures were made. Coccal forms predominated, like *Mycobacterium pulviforme* described by MARCHOUX in 1923 ; others were like those of KEDROWSKY and DUVAL on glycerin agar, but more of an orange than a golden yellow. Further subcultures were made on glycerin potato, glycerin broth and glycerin agar (all with 5 per cent. glycerin) and the yellow growths were obtained with all of them. The early growth was mostly of the coccoid forms, later bacillary, when transferred to the glycerin media. They were always acid- and alcohol-fast.

In an addendum the author describes the results of animal inoculation and further subcultures of the organism. Abscesses in the peritoneum, axilla and elsewhere revealed the same organism, but smears from the internal viscera were negative. Portions of the tissues have been sent to Dr. Torres and to other laboratories for examination and experimental work. A coloured plate shows well the growth and its characters. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. & MIRANDA, R. N. Poderá o carrapato transmitir a lepra? Mais quatro amostras de culturas de bacilos acido-alcool resistentes obtidas de carrapatos (2 de "Amblyomma cajennense" e 2 de "Boophilus microplus") infectados em leprosos do Paraná. 3a Nota. [**Is the Tick a Transmitter of Leprosy?**] *Mem. Inst. Oswaldo Cruz.* 1942, v. 37, No. 3, 391–425, 23 figs. [19 refs.] English summary.

The subtitle of this article—the material on which it is based—is "four more specimens of culture of acid- and alcohol-fast organisms obtained from *Amblyomma cajennense* and *Boophilus microplus*, infected from lepers in Paraná."

The account is very detailed, but the upshot of it is that Dr. Miranda, who is head of the São Roque leprosarium, allowed a number of ticks to feed on lepers and then sent the insects to Professor de Souza-Araujo. On examining them the latter found that *Amblyomma cajennense* and *Boophilus microplus* could infect themselves from the lepers and that the organisms thus taken in would grow on Löwenstein's medium. A study of these cultures is now proceeding and animal inoculations into white rats are to be carried out. These ticks, it was observed, can pass from one host to another and continue their feeding, so that organisms acquired from one host may be transmitted directly to a second. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. Infecção espontânea e experimental de Hematófagos (Ixodídeos, Triatomídeos, Culicídeos, Hirudíneos, Pediculídeos e Cimicídeos) em leprosos. Possibilidade de serem eles vectores ou transmissores da lepra. [**Natural and Experimental Infection of Blood-sucking Insects and Leeches from Lepers.**] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 447–84, 21 figs. [30 refs.] English summary.

The author quotes from the reports of other investigators and then proceeds to relate the results of his own experimental work in testing whether the insects in question become infected after being allowed to feed on lepers. Details are given of each set of experiments and for general information these may be summarized as follows:—

Ticks.—*Amblyomma cajennense* and *Boophilus microplus* caught feeding on lepers were found to contain bacilli acid- and alcohol-fast, Hansen's organism, but growth of them was not obtained. Only female ticks were found thus infected.

Triatoma.—*T. infestans* and *Panstrongylus megistus* [*T. megista*] were found naturally infected. Experiments were carried out with these and with *Rhodnius prolixus* and other Reduviids, and it was found that the larvae of *T. infestans* do not insert the proboscis deeply into the skin, whereas the nymph and adult penetrate more deeply and infect themselves. Some writers have affirmed that bacillaemia occurs in lepers only at the time when there is fever, but this appears to be erroneous.

Culicidae.—*Psorophora ciliata*, *P. varipes*, *P. ferox* and *Aëdes crinifer* were found to have ingested many bacilli after feeding on a leper.

Leeches.—Diametrically opposing opinions have been stated as to the infectivity of water leeches; some have stated that after sucking the blood of lepers no bacilli can be found, others that they are to be seen every time. VAN BREUSEGHEM in 1937 [this *Bulletin*, 1937, v. 34, 905] examined 165 specimens taken from Lake Egoba in the Congo, much frequented by lepers, and found the organism in three of them, very many in one. The author convinced himself that leeches do ingest the organisms and excrete them in their faeces.

Cimicidae.—GOODHUE and others have reported finding these bacilli in *Cimex lectularius* which had fed on lepers, but the author did not find any positive among specimens collected in the beds of lepers and sent to him for examination. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. Verificação, em condições naturais, da infecção de mais três Hematófagos (*Anophelineos, Flebótomos* e *Simulídeos*) em leprosos. [**Natural Infection of Three more Blood-sucking Insects from Lepers.**] *Mem. Inst. Oswaldo Cruz.* 1943, Oct., v. 39, No. 2, 167–76, 14 figs. English summary.

Ten lepers, all L3, were selected and insects which bit them were captured and examined. The author had previously demonstrated infection by acid- and alcohol-fast bacilli in the body louse and in the nymph of the tick *Amblyomma cajennense,* and these findings he was able to confirm, but he also found that two species of *Anopheles*—*A. albitarsis* and *A. tarsimaculatus*—were heavily infected, that *Phlebotomus intermedius* showed the bacilli in both the proboscis and the stomach ; they were present also in smears made from two specimens of *Simulium* (*S. pertinax* ?). *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. Culturas de bacilos ácido-álcool resistentes isolados de hematófagos infectados em leprosos. Evidências de se tratar do bacilo de Hansen. [**Culture of Acid- and Alcohol-fast Organisms from Blood-sucking Insects which have fed on Lepers. Evidence that these are Cultures of Hansen's Bacillus.**] *Mem. Inst. Oswaldo Cruz.* 1944, Feb., v. 40, No. 1, 9–31, 20 coloured figs. on 1 pl. & 40 text figs. English summary.

The author has made a series of cultures primarily from human leprosy lesions and secondarily from the blood-sucking insects which have fed on leper patients, or from animals (guineapigs and white rats) inoculated with the primary cultures. He has now succeeded in obtaining 7 cultures : (1) The primary one direct from a leper (he obtained subcultures from this by inoculation of guineapigs and white rats). (2) Two isolated from *Amblyomma cajennense* which had been allowed to feed on lepers ; two from *Boophilus microplus* similarly obtained. (3) Two from Triatomidae, *T. infestans* and *Panstrongylus megistus.* The characters of the growth have already been described [above] and they are well shown in a coloured plate of 20 figures. Penicillin, which acts so potently on some Gram-positive bacteria, has no effect on this organism. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. Verificação da infecção de moscas da familia *Tachinidae* pela *Empusa* Cohn 1855. Essas moscas, sugando ulceras lepróticas, se infestaram com o bacilo de Hansen. [**Infection of *Tachinidae* by *Empusa* and Hansen's Bacillus.**] *Mem. Inst. Oswaldo Cruz.* 1944, Aug., v. 41, No. 1, 200–203, 3 figs. English summary (8 lines).

On an ulcerous lesion of the left external malleolus of a leper a large number of flies were feeding. Some were captured and were diagnosed as *Tachinidae* ; they were too few for the species to be determined, but it was thought that they were of several species. They were all dead by the following day and in their intestines were acid- and alcohol-fast bacteria mixed with the fungus *Empusa.* Cultures of the bacteria on Löwenstein's medium were attempted, but time was allowed to elapse and they were overlooked. The work is to be taken up again. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C., MARIANO, J. & DE OLIVEIRA CASTRO, G. M. Tentativas de transmissão da lepra ao homem, por meio de Triatomídeos infectados em doentes lepromatosos. (Nota prévia). [**Attempts to transmit Leprosy to Man by Triatomidae which had fed on Lepers.**] *Mem. Inst. Oswaldo Cruz.* 1944, Dec., v. 41, No. 3, 495–505, 4 figs. English summary.

Professor de Souza-Araujo has already demonstrated [above] that Triatomidae, *T. infestans* and *Panstrongylus megistus*, after feeding on a leper, harboured bacteria which were acid- and alcohol-fast and could be grown on Löwenstein's medium. With his fellow-workers he has now studied whether these insects, thus infected, can later infect human beings. To test this they bred *T. infestans* and *P. megistus* in the laboratory till they were sufficiently developed to bite man, when they were allowed to feed on burned-out leprosy patients. Five months later a few acid-fast bacilli could be seen in the lymph lying under the site of the bite—not a very satisfactory result so far, but the patients are being kept under observation for further study. [Not a very satisfying paper; if the disease does develop, it may be looked upon as a lighting-up of a condition believed to be burned-out, and if it does not the question arises whether a burned-out case is re-infectible.] *H. Harold Scott.*

DE OLIVEIRA CASTRO, G. M. & MARIANO, J. Transporte e inoculação de bacilos da lepra pelos mosquitos. [**Mosquitoes and Leprosy Transmission.**] *Mem. Inst. Oswaldo Cruz.* 1944, Dec., v. 41, No. 3, 511–24, 3 figs. & 2 coloured pls.

It is known that after mosquitoes have bitten a leper the bacteria can be demonstrated in the alimentary canal of the insect, but whether the mosquito can inoculate a healthy subject is not known.

Volunteers having allowed themselves to be bitten by such mosquitoes were examined after intervals varying from 14 to 31 days. Lymph was withdrawn from the site of the bite; bacteria were not found, but sections of the skin revealed a perivascular infiltration of the superficial plexuses of the skin, no apparent change in the epidermis, but acid- and alcohol-fast bacteria were seen in the connective tissue spaces, not in the cytoplasm as is usual in leprosy. Whether the mosquitoes really infect at the time of biting, or whether the negative, burned-out cases (on whom these tests were tried) are not really "abacilliferous" but "paucibacilliferous" [*i.e.* still harbour a few organisms] or, thirdly, whether the bite has set up a *locus minoris resistentiae* with consequent multiplication of a few local pre-existing organisms, which of these three hypotheses is the correct interpretation of the findings remains to be proved. *H. Harold Scott.*

IBARRA PEREZ, R. & GONZALEZ PRENDES, M. A. Síntomas iniciales de la lepra. [**Earliest Symptoms of Leprosy.**] *Rev. Sifilografía, Leprología y Dermatología.* Marianao, Cuba. 1945, June, v. 2, No. 2, 108–11.

Between the time of inoculation by Hansen's organism and the appearance of symptoms there is usually a long silent period. In children separated from their parents the interval ranges up to five years. Though the number of bacilli in a lesion is large, its presence may pass unperceived. Reports of the incubation period between inoculation and appearance of symptoms, as, for example, infection of a wound, a surgical operation, by use of a leper's razor, and so on, are not conclusive because the subjects were all living with lepers or in contact with them and might have been infected earlier, or at any time.

The authors have noted the initial lesions, that is the first signs observed by patients, in 760 cases, and in a list they give the numbers and percentages of the first symptoms. Erythematous maculae were given, as the first seen, by

139 (18·3 per cent.), achromic maculae in 104 (13·7), dark macular in 45 (6), *i.e.* macular lesions in 288, or 37·9 per cent.; areas of anaesthesia in 71 (9·34), infiltration of the ears in 66 (8·7), systemic disturbance with fever in 49 (6·44), rhinitis in 33 (4·33). These are the chief, and the great majority were first observed in uncovered parts, face, neck, arms, chest and feet. It is noticed that rhinitis comes a good way down the list and the authors are of opinion that the nose is not the portal of entry but the portal of exit. After a period of time, the bacillus, having gained entry, finds its way to the lymph or blood capillaries; if the former the development is slow, if the latter dissemination is more rapid and widespread, and symptoms such as general malaise, headache, fever and rheumatic pains are observed. *H. Harold Scott.*

DE SOUZA-ARAUJO, H. C. A lepra tuberculoide, ou melhor, a lesão tuberculoide na lepra, representa uma fase de transição desta dermatose e não uma forma clinica autônoma. Novos achados bacterioscópicos na linfa subcutânea de leprosos. [**Tuberculoid Lesions not a Distinct Form of Leprosy.**] *Mem. Inst. Oswaldo Cruz.* 1943, Aug., v. 39, No. 1, 77–96, 18 figs. [12 refs.] English summary.

The author brings evidence to show that the division of leprosy into lepromatous, tuberculoid and macular types is an artificial one and rests on no sound basis. In his view all cases are mixed, those regarded as cutaneous have also the nerves to the part affected. Using the Lleras technique of examining the cutaneous lymph [see this *Bulletin*, 1944, v. 41, 220] in so-called tuberculoid cases he found small granules and coccobacillary forms of the organism, acid- and alcohol-fast. Relapses in burned-out cases are due, it is said, to development of these forms of the bacillus. The article contains a series of excellent photographs showing the "tuberculoid form" of leprosy. *H. Harold Scott.*

GONZÁLEZ UREÑA, J. Alopecia leprosa en Mexico. [**Leprotic Alopecia in Mexico.**] *Rev. Sifilografía, Leprología y Dermatología.* Marianao, Cuba. 1945, June, v. 2, No. 2, 73–100, 20 figs.

DE SOUZA-ARAUJO, H. C. Preparo de antígenos (leprolinas Souza-Araujo) de culturas de bacilos ácido-álcool resistentes isolados de leprosos. Seu emprêgo intradérmico, comparativamente com o da lepromina, e subcutâneo ou intravenoso como tentativa terapêutica. [**Preparation of Souza-Araujo Leprolins: Cultures of Acid- and Alcohol-fast Organisms obtained from Lepers.**] *Mem. Inst. Oswaldo Cruz.* 1943, Dec., v. 39, No. 3, 349–55, 4 figs. English summary.

The technique for preparing the antigens is to extirpate a florid leproma rich in Hansen's bacilli, emulsify it and homogenize it by placing it in an electric shaker for 10 days and nights; then add liquid phenol to 0·5 per cent. of the volume and, after testing it for sterility, add 0·5 per cent. phenol to 10 per cent. of the volume; it is then put up in ampoules. The skin reactions to these are the same as those with lepromin. These antigens are being tested as to their value (*a*) in diagnosis and (*b*) in treatment, and results will be promulgated later. The author kindly offers to place his products—there are five of them—at the disposal of any leprologist who wishes to test them. *H. Harold Scott.*

MARIANO, J. Resultados do emprêgo das leprolinas "Souza-Araujo" comparativamente com o da lepromina. [**Comparison of Leprolins "Souza-Araujo" with Lepromin (as regards Intradermal Tests).**] *Mem. Inst. Oswaldo Cruz.* 1944, Feb., v. 40, No. 1, 101–19, 1 fig.

Five leprolins were obtained: one from a leper, one from a guineapig inoculated with the primary leper culture, two from cultures from *Amblyomma*

cajennense, and one from *Boophilus microplus*. These were inoculated in amounts of 0·2 cc. and compared with a similar inoculation of lepromin. The tests were carried out on 10 groups of lepers, namely, L1 (8 cases), N1 (7), N2 (3), N3 (6), L1N1 (11), L2N2 (3), L1N2 (14), L2N1 (16), L3N1 (19), L3N2 (4). Protocols show the results of four consecutive weekly readings in each case. For purposes of this abstract, the readings (which total more than 2,000) must be grouped. In the L1 cases, leprolin 4 (a strain from *Amblyomma cajennense*) gave reactions identical with lepromin, the others gave differing readings ; in group II, the N1 cases, again leprolin 4 gave the same as lepromin ; in the N2, a group of only three cases, leprolin 2 (the guineapig strain) agreed with lepromin ; among the N3 cases again lepromin and leprolin 2 were in harmony ; in the L1N1 patients, lepromin was negative in all. None of the leprolins agreed with this, but Nos. 3, 4 and 5 all gave 9 negative and 2 positive. Among the three L2N2 patients lepromin agreed with leprolins 1 and 3 (the direct culture and one of the *Amblyomma* cultures), while leprolin 4 (the other *Amblyomma* culture) was negative with all three ; in the L1N2 group, leprolin 3 gave one positive and five negative, whereas lepromin and all the other leprolins gave a negative. [Only six are referred to in the letterpress on this group, although in the table 14 are given.] The L2N1 group we cannot speak of with certainty for the letterpress states that there were 24 patients in the group, whereas the table gives details of 16 only ; anyway none of the leprolin results corresponded exactly with those of lepromin. In the L3N1 group of 19, lepromin was negative in all ; the nearest approach to this was leprolin 3 with two positive. All four of the L3N2 group were negative with lepromin, while of the leprolins, No. 2 was positive in two and negative in two, the other four leprolins being negative in three.

In another group of three contacts lepromin was positive in all, as were also leprolins 2 (guineapig), 3 (an *Amblyomma* strain) and 5 (the *Boophilus* strain). Among another group of children in the Preventorium, leprolins 2, 3, 4 and 5 gave the same total results, two positive, four negative, as lepromin, but the individual readings differed.

Bringing these results together, the author in his summary states that in the nervous cases lepromin gave a positive in 13 and negative in 3, while leprolin 2 was positive in 12 and negative in 4 ; in lepromatous patients lepromin was positive in three, negative in 72 ; the leprolin most nearly approximating to this was No. 3, with 16 positive and 59 negative.

To obtain a closer comparison it is thought necessary to make a fresh series of tests with graded doses of the leprolins ; the author concludes that the leprolins " can play a fundamental part in the prognosis of leprosy since the reactions enable evaluation to be made of the defensive powers of an individual patient." *H. Harold Scott.*

MIRANDA, R. N. Resultados comparativos das reações intra-dermicas com antígenos de bacilos ácido-alcool-resistentes (Leprolinas " Souza-Araujo ") e emulsão de lepromas (Lepromina). (Primeira nota). [**Comparison of the Results of Intradermal Injection of " Souza-Araujo " Leprolins and Lepromin. (First Communication).**] *Mem. Inst. Oswaldo Cruz.* 1944, Aug., v. 41, No. 1, 195–200. English summary.

The author carried out tests similar to the above, on 20 lepers, 17 males, 3 females, 13 lepromatous and 7 nervous or tuberculoid types, at the São Roque Colonial Hospital, Paraná State, Brazil. He obtained his samples of the leprolins from Professor de Souza-Araujo. Readings were taken after 48 hours and at the end of each of the first 4 weeks. The results were, in general, in fairly close agreement with those of de Souza-Araujo, namely, positive in

the nervous, negative in the lepromatous cases. As a rule no constitutional disturbance resulted from the inoculation, but many of the patients showed a scar at the site of inoculation, indicating local destruction of tissue, although there was no preceding ulceration. In one the lepromatous lesions tended to become confluent after the inoculation. *H. Harold Scott.*

MOM, A. M. & BASOMBRIO, G. Estudios de reactibilidad cutanea en lepra, VII. Las intradermorreacciones provocadas por la lepromina y el 2–4 dinitroclorobenceno. [**Skin Reactions in Leprosy. VII. Intradermo-reactions due to Lepromin and 2–4 Dinitrochlorobenzene.**] *Rev. Argentina Dermatosifilologia.* 1945, June, v. 29, No. 2, 120–28, 10 figs. [26 refs.] English summary.

The authors have previously reported their results with these antigens [see this *Bulletin*, 1944, v. 41, 1049] and now put on record further tests, using the same technique as before. The reaction is read at 48 hours (the early reaction), when, if it is positive, the redness and oedema are not less than 10 mm. in diameter ; and at the third week (the later or delayed reaction) when there is a nodule not less than 5 mm. in diameter, with or without softening or central ulceration. The nodular, or late, reaction begins to show itself at the end of the first week and progresses for a fortnight. It may last for 6 weeks or more and may, but does not always, leave a scar. Those are classed as doubtful in which the early reaction is between 5 and 10 mm., and the late 3–5 mm. in diameter.

Tests were performed on 223 persons : 71 lepers with lepromata, 90 with tuberculoid leprosy, 21 "non-characteristic" lepers (182 lepers in all), 15 apparently healthy persons living with lepromatous cases, and 26 healthy subjects having, as far as was known, no contact with lepers.

In the 71 lepromatous patients the reactions were mostly negative with both antigens, early and late. The tests agreed in 69 in the early reactions and disagreed in two ; with the late reaction both were the same in all 71.

In the 90 tuberculoid cases the reactions were positive in the majority and were concordant. With the early reaction there was agreement in 75, non-agreement in 15 ; with the late reaction the respective figures were 81 and 9.

In the 21 non-characteristic, simple inflammatory form, 19 gave the same reactions with both antigens at the early reading, two were discordant, whereas at the late reading 17 were in harmony, four were not.

Of the 15 apparently healthy subjects living with lepers, in the early reaction 12 were in harmony, three were discordant ; in the late all 15 gave the same reactions to both antigens. Lastly, of the 26 healthy subjects, the early reaction was concordant in all but one (dinitrochlorobenzene negative, lepromin positive) ; the late reaction was concordant in 24, discordant in two (both lepromin positive, dinitrochlorobenzene negative).

The histological changes set up by the intradermo-reaction to both these antigens were studied by staining biopsy specimens at the end of 24 and of 48 hours, and at the 4th, 8th, 16th and 30th days ; these are shown by a series of well-produced photomicrographs. *H. Harold Scott.*

DHARMENDRA & RAJAGOPALAN, S. **Some Aspects of Chemotherapy in Leprosy.** *Leprosy in India.* 1945, July, v. 17, No. 3, 77–84. [53 refs.]

Owing to the limitations of the hydnocarpus oil treatment of leprosy, the search for a more effective treatment for advanced cases is necessary if early control of the disease is to be obtained. Drugs of the sulphonamide group have not proved to be of much use, but sulphone compounds promise better in

mycobacterial diseases such as tuberculosis and leprosy. Of these, "promin" has already been favourably reported on by American workers, and "diasone" has been found to have some prohibitive effect in tuberculosis in guineapigs, as has "promizole"; they are therefore worthy of careful trial in leprosy. The high lipoid content of the mycobacteria increases their resisting powers, so any change in the constitution of sulphonamides which makes them lipophilic may be expected to increase their effects. Efforts already made in this direction by other workers are summarized in this article, which should be read by those interested in this line of research. The Leprosy Department of the School of Tropical Medicine, Calcutta, hopes in due course to report on such studies.

L. Rogers.

CHORINE, V. Traitement des lésions oculaires de la lèpre. [**Treatment of the Ocular Lesions of Leprosy.**] *Bull. Acad. Méd.* 1945, v. 129, Nos. 13, 14 & 15, 230–32.

This note records promising results in the treatment of the difficult eye lesions of leprosy by local injections of sulphonamides. In order to obtain the sulphonamide in solution, 60 gm. of acetamide and 15 gm. of sulphanilamide are made up to 100 cc. with distilled water. This solution diffuses slowly through the tissues when injected around a lesion of the skin, and causes its gradual disappearance. In the case of eye lesions, periorbital injections are made of two to four injections of 7–10 cc. of the solution, but, owing to the severe pain they cause, local anaesthesia is first induced by the use of 1 to 2 per cent. of stovaine. No complications have been noted after weekly injections at different sites. Thirteen eye cases (keratitis, irido-cyclitis, etc.) have been treated, but five are too recent to be reported on. Of the other eight cases, in four vision has recovered to normal, and in one after only nine injections it has improved. In the other three, blindness of several months' or years' duration failed to respond to the treatment, but pain in the eyes has been reduced. In eye cases photophobia has been cured and rapid retrogression of keratitis has been observed. Irido-cyclitis is less quickly influenced, but is eventually quieted. Moreover, nasal infections have been cured in all such cases. In the case of localized dermal nodules the results are also very favourable. [Further trials of this promising method of treatment will be awaited with interest.]

L. Rogers.

LEPROSY REVIEW. 1945, Aug., v. 16, No. 1, 4–12, 1 fig. **New Drugs for Trial in Leprosy.**

DAVEY, T. F. **Leprosy Control in Owerri Province of Nigeria.** *Leprosy Review.* 1945, Aug., v. 16, No. 1, 21–30.

This report deals with the sixth year's work of this great leprosy colony [see this *Bulletin*, 1944, v. 41, 53 for previous report]. Of the two million inhabitants of the Owerri Province it is estimated that not far short of 75,000 suffer from leprosy, yet two of the six divisions are still untouched in spite of 18,554 patients having been brought within the scope of the work. It is impossible to isolate all the infective cases, but working on a clan basis, with the cordial co-operation of the people themselves, 27 leper villages have been built to accommodate 1,807 of the most highly infective patients, and six more are waiting development. In addition 1,179 patients are living in the central settlement, nearly all infective or requiring hospital treatment. Over forty out-patient clinics supply treatment to earlier cases. Repeated surveys allow newly developing cases to be discovered and treated while still in an amenable

stage. Nurses are given eighteen months' training, and 130 are already at work in the clinics and villages. Over 1,100 child patients are being cared for; the majority of these are maintained by B.E.L.R.A.'s child adoption scheme financed by special subscriptions given largely by British children. Physical training, boy scout activities and various social amenities are encouraged or supplied. Twenty leprosy inspectors work in different parts of the province; they include in their duties the examination of contacts of known infective leprosy cases, especially children, for early signs of infection. Already, in the case of six clans, the present progress amounts to an approximate isolation with treatment of all infectious cases, together with treatment of all the earlier cases. In these areas progressive diminution in the incidence of leprosy may be expected. Thus the practicability of effecting a rapid reduction of leprosy in Nigeria with sufficient staff and funds has been demonstrated, and it is this success which has led the Nigerian Government to take over the work with a view greatly to extend its scope. *L. Rogers.*

SEN, J. B. **Purulia Naba-Kustha-Nibas (Balarampore). A New Line of Attack on the Leprosy Problem.** *Leprosy in India.* 1945, Apr., v. 17, No. 2, 43–7.

This is an interesting account of a trial of a new and promising method of dealing with the serious problem of the wandering begging lepers, who help to spread the disease in India, though to a limited extent because many of them are of an advanced, crippled, little infective nerve type. The term Naba-Kustha Nibas means the New Leper Home. The need for its foundation was demonstrated by the popularity of the large leprosy institution at Purulia, which attracted hundreds of begging lepers from other parts of India, who could not be admitted to the older institution. It was originally intended as a night shelter, but owing to the generosity of the people of Purulia, with the aid of grants from local government bodies, it has been developed into a home in which the unfortunate patients are lodged and fed four miles out of the town. It was found that many of them were fairly well-to-do persons, who had been driven out by their relatives, and some of them have recovered part of their property and given it for the support of the new home, which now contains some 300 patients.

Many of them do agricultural work to help in the feeding of the patients, and others are employed on weaving or making pottery, etc. Thus new hope and interest in life is provided, and the patients are regarded as guests of the town. This plan is worthy of imitation by the numerous cities of India in which the begging leper is a nuisance, discreditable to those who neglect the care of their afflicted relatives. [Much money has for long been expended in India in providing homes for keeping alive maimed and diseased cattle, which Hindus are prohibited from putting out of their misery, because cows are sacred in their eyes. The above is, however, the first attempt the reviewer can recall of the people of India raising funds for providing homes for their own leper outcasts; hence its importance.] *L. Rogers.*

LINHARES, Herminio. Possibilidades de transmissão e vias de inoculação da lepra murina em ratos e outros animais. [**Possible Routes of Transmission of Rat Leprosy in Rats and other Animals.**] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 321–51. [101 refs.] English summary.

In the first part of this contribution the author mentions the many possible ways by which rats may become infected with the Stéfansky organism, giving quotations from the literature dealing with the experiments connected therewith. He deals with: (1) Insect transmission by *Lucilia caesar, Calliphora vomitoria, Musca domestica, Haematopinus spinulosus, Ctenocephalus serraticeps,*

Laelaps echidninus, *Polyplax spinulosa* and others, showing how the organism has been found in them and attempts have been made to cultivate them, usually with barren results. The insects certainly contain acid-fast bacilli and if the rats devour a large number of them they may become infected, but this is not insect transmission in the usual and accepted sense ; it would come rather under the next heading. (2) Infection by the alimentary route. Rats fight, and eat their dead opponents, and may thus become infected. Feeding rats with rat leproma tissue rich in bacilli is followed by widespread infection, in glands, liver, lungs, spleen and kidneys in 24 weeks onwards. (3) Cutaneous infection ; scratches, wounds and bites are frequent among rats owing to their fighting and the organisms can easily enter thereby. (4) Congenital transmission finds no evidential support. (5) Intranasal infiltration. Some have reported finding the organism in the nasal secretion, others have tried to produce the disease by experimental instillation, but without result. A certain species, the camondongo, has shown pulmonary infection from this, but the ordinary rat is less susceptible and is rarely, if ever, thus infected. Other methods, such as intradermal, intracardiac, intravenous and intracerebral, are of academic interest only. Genital infection occurs experimentally and the bacteria can traverse the vaginal mucous membrane and in the end produce generalized disease.

In the second part quotations from the literature tell of the successful results of inoculation into guineapigs, rabbits, camondongos, *Macacus rhesus*, opossums, fowls and pigeons by various routes. The author ends by giving an account of his own attempts to infect fowls and pigeons. He was able to infect the former by feeding and also by intramuscular and intraperitoneal injection ; the latter by intramuscular or intravenous inoculation.

H. Harold Scott.

HELMINTHIASIS.

SARMENTO, A. Nota sôbre um foco de bilharziose vesical em Angola. [**A Focus of Vesical Schistosomiasis in Angola.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 375–80, 1 folding map.

In March 1939 the author made a health survey of the Province of Bié to determine the degree of endemicity of *Schistosomiasis haematobium* there. Several foci were found, the chief being Catote, Chibia and Ganguelas, the last extending to Cuchi. A spot map indicates some of the foci. From 1936 to 1941 registered cases of this disease in Angola have numbered 1,888, 1,857, 1,344, 1,858 and 1,792 respectively.

In the Cuchi area the author found many cases ; several of the patients were passing visible blood. The total numbers examined are not stated, but the percentages infested (shown by microscopical examination of the urine) were : Children 60·2, adults 21·5, or, both together, 54·5. Figures for other parts of Africa are given for comparison : Environs of Dakar 38·8, Toma (French Guinea) 54·5, Kiné (French Guinea) 9, Liberia 38–56, Sierra Leone 13–26, Victoria Nyassa [*sic* ? Nyanza] 50, Mombasa 83, Southern Rhodesia 50, Pretoria 31. *Bulinus* (species not stated) was found on the borders of streams and rivers, Bissombo, Cuchi, Luassenha and Musanga. Though the disease is not often the cause of death, it reduces the working capacity of many by the resultant anaemia and debility and by lowering the power of resistance to other diseases.

H. Harold Scott.

MAGALHÃES, A., COUTINHO, B., GOUVÊA, L., LUCENA, D. & IGNACIO, L. Estudos sobre a Esquistosomose em Pernambuco, Brasil. [**Schistosomiasis in Pernambuco.**] *Mem. Inst. Oswaldo Cruz.* 1940, Jan.–June, v. 35, No. 1, 205–83, 15 graphs & 29 pls.

This article is divided into three parts: the first treats of the epidemiology of *Schistosomiasis mansoni* in Pernambuco and is mainly of local interest. Sketch maps give the situation of Pontezinha and Vitoria, places in the State where the disease prevails. These maps show the water collections and general lie of the land and, by varied shading, the degrees of incidence. In some parts of Pontezinha this is as high as 39 per cent. and the incidence rises with age to the 16–20 years group, when it reaches 37·8 per cent.; the ratio of males to females affected is almost 3 : 2 (21·4 and 14·7 respectively). The highest age incidence in Vitoria is in the 10–15 years age-group, when it reaches 56·9 per cent.; the 16–20 years group has an incidence of 52·2 per cent. Here, however, males are proportionately less affected than females, 36·9 to 41·4 per cent. This part is embellished with 14 photographs illustrating the local terrain.

Part II is of more general importance and will be studied by all those practising in countries where this infestation is common. It is a detailed histological study of the lesions in the various organs: the rectum, liver, lungs, pancreas, spleen and glands. Forty-three beautifully reproduced photomicrographs illustrate the histological changes set up. Part III gives an account of nine patients with reference particularly to the condition of the blood and the spleen. The blood changes strongly resembled those of chronic splenic anaemia. In four cases splenectomy was carried out, but the results were not good on the whole. Such changes as there were tended to better regulation in the proportions of the cellular elements. One patient improved; one was lost sight of, a third showed little if any change in the blood condition and died; the fourth left hospital no better. *H. Harold Scott.*

MAGALHÃES, B. F. & DIAS, C. B. Esquistossomose de Manson—Estudos. [*Schistosomiasis mansoni* (**in Brazil**).] *Mem. Inst. Oswaldo Cruz.* 1944, Dec., v. 41, No. 3, 363–446, 1 map, 1 coloured pl. & numerous figs. [234 refs.]

This study is divided into four chapters. The first is a short one of 4 pages, of historical interest, discussing the question whether rectal schistosomiasis was brought originally by slaves from Africa or was autochthonous. The authors conclude that the evidence in favour of introduction by the negroes is not strong, whereas there is much to support the view that the disease existed before the time of their introduction. The snails were present, it is said, before slaves were being brought, and VON MARTIUS has told that among the Indians (the aborigines) there was a disease characterized by a chronic hepatitis (though other organs were also affected) and often by enlargement of the spleen, "haemorrhoids" and diarrhoea.

The second chapter of just over 3 pages is little more than a list of Brazilian workers who have been engaged on the subject. Chapter III, occupying most of the thesis (56 pages) is concerned with antimonial treatment of schistosomiasis, its dangers and the fatalities associated with its use, together with experimental work on 27 patients treated with it and on 7 dogs to which the drug was given in toxic doses. Photomicrographs, one in colour, show well the histological changes in the liver and heart. The effects on the heart form the main subject of this study. Antimony seems to act on the capillary walls, leading to such a degree of dilatation that there is a diminution in the volume

of the circulating blood, associated with inadequate supply by the coronary vessels to the heart itself. The work on the effects of antimony on the heart is illustrated by over 300 electrocardiographic tracings; a few words of letterpress are given on each [which is well, for unfortunately the tracings are so reduced in reproduction that they are illegible even with a magnifying glass].

The final chapter is a brief one of two pages devoted to consideration of whether or not there is a cardiac form of *Schistosomiasis mansoni*. There have been very few records of myocardial lesions in this disease [except, presumably, after antimonial treatment] and of the many cases studied, and particularly among the 22 autopsies by the authors, though the ova have been found in the ileum, caecum, rectum, liver, lungs, ovaries, testes, bladder and elsewhere, there is no record of their localization in the heart; it is highly improbable, therefore, that there is a cardiac form of the disease. *H. Harold Scott.*

SCHWETZ, J. & DARTEVELLE, E. Sur la faune malacologique comparative des lacs Albert, Edouard et Kivu. [**The Snails of Lakes Albert, Edward, and Kivu.**] *C. R. Soc. Biol.* 1945, Jan., v. 139, Nos. 1–2, 69–72.

CAIRO: MINISTRY OF PUBLIC HEALTH. MEDICAL AFFAIRS. **First Annual Report. Bilharzia Snail Destruction Section 1942** [BARLOW, C. H. (Expert) & ABDEL AZIM, M. (Director)]. 21 pp., 3 maps (1 folding) & 5 figs. (3 on 2 pls.). 1945. Bulaq, Cairo: Govt. Press.

In Fayoum Province, 4,720 kilometres of irrigation canals and drains support snail populations of the bilharzia carrier *Bulinus truncatus*. These are most abundant in the branches of Bahr Yussef in the central part of the Province, where the sluggish flow and muddy bottoms of the waterways provide the most favourable environment.

The system of bilharzia control, initiated in 1942, is based on two main methods. (1) Mechanical clearance of small streams not exceeding $1\frac{1}{4}$ metres in width by means of standardized hand nets, which also serve as a measurement of snail intensity and as a guide to the progress of the control work. A coarse mesh net removes large snails, vegetation, litter and a top layer of mud, after a preliminary loosening and clearance of vegetation by hoeing and manual work. This is followed up by dipping with fine mesh nets which removes small snails, egg-nests, ooze and fine plant refuse. (2) Chemical treatment of larger canals with copper sulphate. Preliminary laboratory work showed that 7 parts per million of this salt, acting for 48 hours, was an effective killing agent, but in field practice concentrations of 15–50 parts per million were required and varied according to the amount of vegetation and silt in the canals and to the length of time available for the treatment, which consists simply of dragging a bag of $CuSO_4$ through the water at a speed calculated to give the required concentration.

In the laboratory attached to the Snail Destruction Section, problems arising in the course of the field work are studied, routine analyses of water and snails are carried out and the personnel is trained. Studies on the life span and growth of *B. truncatus* and *P. boissyi* showed that *Bulinus* lived for 11 months during which time it attained an average length of 12·5 mm. *P. boissyi* lived 18 months and grew to an average diameter of 13·2 mm. It is concluded that these species do not live longer than two years.

The annual emptying of irrigation channels for a period of 40 days causes the desiccation of the snails, but does not kill them. In laboratory experiments *P. boissyi* revived after 10 months desiccation in mud and *B. truncatus* after 12 months.

Self-fertilization in *P. boissyi* was demonstrated in the laboratory and was successfully continued through six generations. *J. J. C. Buckley.*

JANSEN, G. Observações sôbre o combate à Esquistosomose humana em Pernambuco, no município de Catende. Indice de infestação em Australorbis e emprêgo da cal extinta e do sulfato de cobre no combate aos moluscos. [**Campaign against *Schistosoma mansoni* in Calende, Pernambuco. Incidence of Infestation of *Australorbis centrimetralis* and their Extinction by Slaked Lime and Copper Sulphate.**] *Mem. Inst. Oswaldo Cruz.* 1943, Dec., v. 39, No. 3, 335–47, 14 figs. English summary (10 lines).

The district in question is heavily infested ; examination in one part revealed an incidence of 50 per cent. harbouring *S. mansoni*. The vector is *Australorbis centrimetralis* which abounds in the brooks and irrigation canals where the water is more still than in the flowing streams and rivers. The inhabitants frequent these places for bathing, for washing their clothes, for fishing, for collecting sand, and other purposes, and in parts, the Panelas River for example, those dwelling on the banks have no proper sanitary arrangements and discharge their excreta directly into the water.

Experiments were carried out to test the efficacy of various strengths of slaked lime and of copper sulphate for killing the snail. Boxes 10×10×4 cm. with a gauze front were placed in the water and in them a known number of the snails. The lime or copper was then added to the water and after an interval of 24 hours the boxes were opened and the proportion of snails killed was determined. One hundred per cent. fatality was found to result from the use of slaked lime in a strength of 4–5 per thousand, and the cost of this was only 25–30 centavos per kilo.

Tests with sulphate of copper showed that in a strength of 0·5 per thousand, 18 per cent. of the snails were killed in 48 hours ; with 1·0 per thousand [by a mistake this is stated as 0·1 $^0/_{00}$] 43 per cent. were killed ; with a strength of 2 per thousand all were killed. The copper salt is more expensive than the lime and less easily obtained. *H. Harold Scott.*

THOMAS, H. M., Jr. & GAGE, D. P. **Symptomatology of Early Schistosomiasis Japonica.** *Bull. U.S. Army Med. Dept.* 1945, Aug., v. 4, No. 2, 197–202.

The authors give an account of 41 cases of schistosomiasis due to *S. japonicum*, contracted during the invasion of Leyte Island in 1944. The snail which serves as intermediate host in that area is *Schistosomophora hydrobiopsis*. The date of exposure could be determined in many cases ; in some the infection was contracted during a single short swim in a river.

The symptomatology of the early stages of this infection is not well known. In four cases there was a history of burning and itching sensations immediately after exposure. In others the first symptoms appeared at the end of the third or fourth week, but these might occur at any time up to the ninth week.

" On admission to hospital, the chief complaints were chills, fever, sweats, headache, backache, nonproductive cough, abdominal discomfort, and profound anorexia. Urticaria accompanied these symptoms in one-quarter of the cases and diarrhea in one-fifth. Symptoms in order of frequency in 41 cases were : headache 35, anorexia 30, cough 30, chills 23, abdominal cramps 16, backache 12, diarrhea 8, itching 7. Fever was present to some degree in all cases. In two cases symptoms of central nervous system origin were present. Other symptoms sometimes seen are stiffness of neck muscles, chest pain, abdominal cramps, and loss of weight. Symptoms and signs of acute appendicitis have been reported in other series."

Urticaria was seen in 10 cases. In two, radiographs of the lungs showed small areas of infiltration. The most constant finding was enlargement of the liver (30 cases) with tenderness; the spleen was enlarged in nine. The leucocyte counts were characteristic; there was leucocytosis to 50,000, with eosinophilia from 15 per cent. at first to as much as 90 per cent. later. Schistosome eggs were first found in the stools from 6½ to 10 weeks after first exposure.

The central nervous system was apparently involved in two cases (and in one other not in this series). The patients had weakness of the arms and spasticity of the legs, with increased reflexes. Sensation was normal. One patient was disorientated (he also showed signs typical of occlusion of end arteries in the toes); another was admitted in coma, and died. One other patient died suddenly shortly after the ninth dose of tartar emetic; he also had amoebiasis, but it is possible that the fatal outcome was precipitated by the drug. Fouadin and tartar emetic were the drugs used in this series; symptoms were promptly relieved, but at the time of this report it was too early to assess the final results. Treatment should be given as early as possible.

It is noted that further experience has shown that many persons may show eggs in the stools without having had more than trivial symptoms; severe disease may develop later, unless treatment is given.

"Much weight should be given to a clear history of exposure. If exposure is followed in three or four weeks or less by itching, urticaria, fever, dry cough, and increasing eosinophilia, in the absence of other satisfactory explanation, schistosomiasis may be regarded as probably present. Under these circumstances, even though the diagnosis has not been proved, it may be wise to initiate treatment, using fuadin because of its lower toxicity. Every effort should be made, however, to prove the diagnosis." *Charles Wilcocks.*

BULL. U.S. ARMY MED. DEPT. 1945, Aug., v. 4, No. 2, 178–80. **The Chemotherapy of Schistosomiasis Japonica.**

Very little is known about the results of chemotherapy in *S. japonicum* infections; the drugs in use have been given empirically, without the application of modern pharmacological techniques. The two most largely used are tartar emetic and fouadin. Neostibosan and other pentavalent antimony compounds appear to be relatively ineffectual; anthiomaline has not, apparently, been used, and preliminary experimental work is not encouraging; emetine cannot safely be given in effective doses.

Fouadin and tartar emetic are therefore advised; both may be toxic and both should be injected slowly. Fouadin should be tried first; it is supplied in ampoules of a 6·4 per cent. solution, of which 1·5 cc., 3·5 cc., and 5·0 cc. are injected intramuscularly on three successive days, followed by 5·0 cc. on alternate days to a total of 16 doses.

Tartar emetic contains a much higher proportion of antimony. It should be made up as a 0·5 per cent. solution in 5 per cent. glucose saline (or in saline or distilled water if glucose is not available), and the first dose is 8·0 cc. given intravenously (slowly) to the recumbent patient, who should remain recumbent for one hour afterwards. Subsequent doses (on alternate days) are increased by 4·0 cc. until the dose reaches 28·0 cc. The whole course comprises 15 injections.

If a second course of either of these is necessary, it should not be given until two weeks after the completion of the first. The results of treatment should be checked by stool examinations for fertile eggs. *Charles Wilcocks.*

HUNT, A. R. **Schistosomiasis in Naval Personnel. A Report of Sixteen Cases.** *U.S. Nav. Med. Bull.* 1945, Sept., v. 45, No. 3, 407–19.

The infection was with *Schistosoma japonicum.*

TORRES, C. M. Alterações patológicas do apêndice vermiforme contendo Proglottis de *Taenia* sp. [**Pathological Changes in the Appendix containing a Proglottis of *Taenia* sp.**] *Mem. Inst. Oswaldo Cruz.* 1943, June, v. 38, No. 3, 373–84, 8 figs. [13 refs.] English summary.

CRUZ, W. O. & DE MELLO, R. P. Eliminação urinaria do cloreto de sodio na anemia ancilostomotica. [**Urinary Excretion of Sodium Chloride in Hookworm Anaemia.**] *Mem. Inst. Oswaldo Cruz.* 1944, Oct., v. 41, No. 2, 223–31, 3 figs. & 4 graphs. English summary (4 lines).

The amount of sodium chloride excreted daily in the urine was estimated in six cases of hookworm anaemia over periods of several weeks; during this time the patients were given iron salts for the anaemia but no vermifuge. The haemoglobin in the blood was also measured. The results are shown in tables and charts. In three cases the amount of sodium chloride excreted daily during a control period before treatment with iron was begun was less than 8 gm. It was not correlated with the degree of anaemia, but during treatment it increased in close relation with the increase in the haemoglobin.

The authors cannot explain the diminished excretion of sodium chloride in hookworm anaemia. *J. F. Corson.*

DESPORTES, Camille. Sur *Strongyloides stercoralis* (Bavay 1876) et sur les *Strongyloides* de primates. [*Strongyloides stercoralis* **and the** *Strongyloides* **of Primates.**] *Ann. Parasit. Humaine et Comparée.* 1944–1945, v. 20, Nos. 3–4, 160–90, 3 figs. [Bibliography.]

HODGE, I. G., DENHOFF, E. & VANDER VEER, J. B. **Early Filariasis (Bancrofti) in American Soldiers.** *Amer. J. Med. Sci.* 1945, Aug., v. 210, No. 2, 207–23, 3 charts. [14 refs.]

Sixty-two cases diagnosed as filariasis bancrofti were observed; they could be classified into five groups: I, with genital lesions (47); II, with superficial lymphangitis in the extremities (13); III, with multiple lesions (16); IV, with apparent involvement of the deep lymphatics (1); V, with microfilariae in the blood but without symptoms (1). Cases in Group III were distributed among the other groups according to the first symptom. Notes of illustrative cases are given.

Microfilariae were found in the blood of two out of 266 soldiers, and an adult filaria was found in 1 of 8 lymph glands examined; sections of lymphatic glands showed various degrees of inflammation with infiltration of eosinophil leucocytes.

Regarding eosinophilia as the condition when eosinophils formed 9 per cent. or more of the blood leucocytes, it was present in 18·5 per cent. of 269 soldiers exposed to filarial infection, and in 5·4 per cent. of 240 soldiers not so exposed. The presence of intestinal helminths and of other causes of eosinophilia was excluded by appropriate examinations. Among the 269 who had been exposed to infection, 136 showed signs of filariasis, and of these 24·2 per cent. had eosinophilia, while of the remaining 133, without signs of filariasis, 12·2 per cent. had eosinophilia. In the group of 136 soldiers "with filariasis," 50 had "clinical filariasis" and seven of these had eosinophilia, while of the other 86 with "minimal or doubtful filariasis," 26 had eosinophilia.

An extract of a lymphatic gland was made by digesting it for 12 hours at 37°C. with 0·6 per cent. of pepsin and 0·3 per cent. of HCl, straining, standing, and treating the supernatant fluid with acetone and ether "until a powder was obtained." This was diluted 1 : 20 in normal saline containing 0·4 per

cent. of phenol, and was used for a skin test; 0·1 cc. was injected intradermally and an erythematous weal of 0·5 cm. or more in diameter appearing within 36 hours, was regarded as a positive reaction. Of 11 patients with clinical filariasis seven showed a positive reaction, and all the controls (men not exposed to infection) gave a negative reaction.

The soldiers were exposed to filarial infection for 364 days and typical clinical signs were present in 11 per cent. but the authors think that the rate of infection will eventually prove to have been over 50 per cent. As regards prognosis, the authors think that elephantiasis is due to secondary bacterial infection superimposed on a damaged lymphatic system, and that the risk of the soldiers developing it is slight. The psychosomatic effects of infection were unimportant.

Prophylaxis and treatment are briefly discussed. *J. F. Corson.*

FARINAUD, M. E. Une médication nouvelle : le violet de gentiane et les dérivés du triphénylméthane dans le traitement de l'oxyurose. [**Gentian Violet and Derivatives of Triphenylmethane in the Treatment of** *Enterobius* **Infections.**] *Méd. Trop.* Marseilles. 1944, Sept.–Oct.–Nov.–Dec., v. 4, No. 4, 305–11.

SPRUE.

CAYER, D., RUFFIN, J. M. & PERLZWEIG, W. A. **Vitamin Levels in Sprue.** *Amer. J. Med. Sci.* 1945, Aug., v. 210, No. 2, 200–207, 5 charts. [13 refs.]

Laboratory tests recently devised for determination of the levels of various vitamins have been applied to 12 sprue patients. These were selected on the basis of steatorrhoea, glossitis, macrocytic hyperchromic anaemia, flat glucose tolerance curve and marked weight-loss.

For comparison 25 patients diagnosed clinically as having mild or early deficiency of the B-complex, were selected, and a control group of 30 normal medical students were also studied.

All patients were hospitalized on standard diet, low in vitamins, though adequate in proteins and calories. The control students were permitted to continue normal activities.

Vitamin C was measured in the Evelyn photoelectric colorimeter, using Tillmans' dye and the technique of MINDLIN and BUTLER (*J. Biol. Chem.*, 1938, v. 122, 673). Vitamin A determinations were made by the method of KIMBLE (*J. Lab. & Clin. Med.*, 1939, v. 24, 1055); those of the vitamin B-complex were determined in the urine before and after test doses of thiamin, 1 mgm. i.m., riboflavin after 5 mgm. orally; nicotinic acid amide, 500 mgm. orally; and pyridoxine, 50 mgm. orally. Thiamin was determined at first by the yeast fermentation technique of ATKIN, SCHULTZ and FREY (*J. Biol. Chem.*, 1939, v. 129, 471) later by a modification of the thiochrome method. Riboflavin was measured by the direct fluorometric method of FERREBEE (*J. Clin. Investigation*, 1940, v. 19, 251).

The nicotinic acid load-test was based on the studies of PERLZWEIG, SARETT and MARGOLIS (*J. Amer. Med. Ass.*, 1942, v. 118, 28). The values of F_2 determined fluorometrically paralleled closely those for total nicotinic acid excretion. Pyroxidine was determined calorimetrically by the technique of Scudi *et al.*, including the form hydrolysed by treating with acid.

Vitamin A.—The plasma levels of this vitamin in normal persons showed a distribution and a mean above those of the 25 vitamin-deficient subjects, whilst an even lower figure was determined for sprue.

Carotene.—The general distribution of values for plasma carotene was the same as that for vitamin A.

Vitamin C.—Whilst there were certain individuals in each group whose plasma contained no measurable vitamin C, over half the sprue patients had zero levels and the remainder were below 0·3 mgm. per 100 cc.

Nicotinic acid.—The mean of sprue patients, following administration of 500 mgm. of nicotinic acid, is somewhat higher than that of the deficiency group, but both were considerably lower than normal controls.

Riboflavin.—The mean of patients with sprue was definitely lower than that of patients classified as having a vitamin deficiency, and both were significantly lower than that of the normal control group.

Thiamin.—During the control period of thiamin excretion the distribution of values between the groups was essentially the same as in the case of riboflavin. After intramuscular injection of 1 mgm. of thiamin the means of excretion levels were the same in sprue patients as in those with a vitamin deficiency.

Single deficiencies are rarely, if ever, seen clinically. In sprue anorexia, diarrhoea and interference with absorption determine multiple vitamin deficiencies. This has already been noted, except in the case of nicotinic acid. A possible explanation may lie in the wasting with destruction of body tissues, which would afford a certain amount of nicotinic acid for utilization by the body.

Fats are poorly absorbed in sprue, so that vitamin A and carotene, being fat-soluble, are lost. It might be supposed that the diarrhoea alone is responsible for these low levels, but a comparison with patients having amoebic dysentery shows that this is not the case. In eight patients with amoebic dysentery the mean blood plasma-level of carotene was 141 I.U. as compared with a mean of 50 I.U. in the sprue group, which would suggest that carotene is absorbed in the small intestine. There is likewise a striking difference between carotene and vitamin A levels in sprue as compared with those in pernicious anaemia, in which they are much higher. The striking differences in distribution and means of vitamin A and carotene suggest that these determinations may prove useful as an aid in the diagnosis of sprue and its differentiation from pernicious anaemia.

P. Manson-Bahr.

REWELL, R. E. **A Case of the Sprue Syndrome of Unusual Aetiology.** *Guy's Hosp. Reports.* 1944, v. 93 (v. 23, 4th ser.), Nos. 3/4, 74–8, 2 figs. on 1 pl. [13 refs.]

HAEMATOLOGY.

BROWN, A. **Clinical Haemoglobinometry.** *Glasgow Med. J.* 1945, Aug., v. 26, 39-43. [12 refs.]

The evolution of the technique of clinical haemoglobinometry is reviewed briefly and attention is drawn to the widely varying standards of normality that have been adopted by the earlier workers.

Twenty Sahli haemoglobinometers in general use in a large teaching hospital were checked against an accurate photoelectric method, and readings for 100 per cent. were found to vary from 10·4 to 17 gm. of haemoglobin per cent. This variation was not entirely due to faulty replacements in old apparatus, since "manufacturers appear to have their own ideas with regard to normal haemoglobin levels."

The author deprecates the expression of haemoglobin values as percentages of an arbitrary normal.

[The inaccuracy of many haemoglobinometers in general use is of course now being widely recognized. To some extent this fault is being rectified by the adoption of the specifications for the Haldane instrument recently published by the Medical Research Council's Committee on Haemoglobin Surveys. If haemoglobinometers manufactured according to such specified standards be used, it is difficult to see why the readings should not be expressed as percentages of normal, provided that the equivalent of 100 per cent. in terms of grammes of haemoglobin per 100 cc. of blood is known.] *L. J. Davis.*

MOLE, R. H. **Diurnal and Sampling Variations in the Determination of Haemoglobin.** *J. Physiology.* 1945, June 29, v. 104, No. 1, 1–5.

Various reports of repeated haemoglobin determinations on the same individual have shown considerable variation during the course of a single day. With the object of determining whether such variability is due to sampling and technical errors or to a true diurnal variation, the statistical method of variance analysis has been applied to an examination of certain careful observations previously recorded by McCARTHY and VAN SLYKE (*J. Biol. Chem.*, 1939, v. 128, 567), who used the carbon monoxide capacity method for haemoglobin estimation.

It is concluded that between 9 a.m. and 11 p.m. there was a significant fall in haemoglobin of about 4 per cent., but that there was no significant diurnal change during the working day from 9 a.m. to 5 p.m.

It is also pointed out "that if the diurnal variation is disregarded and included in the random variability, the standard deviation of repeated estimations is 0·54 vol. of carbon monoxide. On the normally accepted basis of twice the standard error, this means that there must be a difference of nearly 8 per cent. between two haemoglobin estimations on one individual before this difference can be attributed with safety to anything but experimental error. In most experimental and clinical work where haemoglobin measurements are used to assess an experimental or therapeutic procedure, there will be at least as big an experimental error as this, against which changes in haemoglobin must be interpreted. The magnitude of sampling and diurnal variations sets a limit, too, beyond which it becomes no longer useful to increase the accuracy of haemoglobinometer readings. The limit is reached when the standard deviation of repeated measurement is about 1% on Haldane's scale." *L. J. Davis.*

BROWN, A. & ANDERSON, A. B. **The Photoelectric Estimation of Haemoglobin.** *Glasgow Med. J.* 1945, Aug., v. 26, 44–9. [21 refs.]

After a short critical review of current methods of haemoglobinometry, a description is given of the author's technique of haemoglobin estimation with a photo-electric colorimeter. The calibration of this instrument by oxygen capacity of the blood is described, and the results obtained on a series of blood samples varying in haemoglobin content from 6·6 to 16·3 gm. per 100 cc. were found to be in very close correlation with those obtained by the direct estimation of oxygen capacity. *L. J. Davis.*

CASE, R. A. M. **Siderocytes in Haemolytic Diseases : a New Index of Severity and Progress.** *J. Path. & Bact.* 1945, Apr., v. 57, No. 2, 271–3. [14 refs.]

Siderocytes are erythrocytes containing fragments of non-haematin iron. They were so named and brought into prominence by GRÜNEBERG (*Nature*, 1941, July 26, 114, & Oct. 18, 469), and have been reported in certain pathological conditions. The author has previously described a new and delicate staining

technique with αα′ dipyridyl and potassium thiocyanate, and has shown that the siderotic granules are eventually extruded, giving an increase of plasma iron, that the cell after extrusion of the granules is susceptible of phagocytosis, and that the onset of a high siderocyte level is accompanied by the appearance of urinary siderotic granules. It was therefore inferred that siderocytes might be increased in haemolytic conditions.

The present communication records siderocyte counts in a variety of conditions, including those characterized by excessive red cell destruction. In normal individuals the siderocyte count was from 0·5 to 0·8 per cent.; in hypochromic microcytic anaemia from 1 to 3 per cent.; whereas in various types of haemolytic anaemia it ranged from 10 to 100 per cent. In untreated pernicious anaemia, counts of 8 to 14 per cent. were recorded, but during the course of therapeutic remission they fall to normal levels.

It is concluded that the siderocyte count may be a sensitive and useful guide in assessing the severity and progress of haemolytic conditions.

L. J. Davis.

WINSOR, T. & BURCH, G. E. **Habitus of Patients with Active Sickle Cell Anemia of Long Duration.** *Arch. Intern. Med.* 1945, July, v. 76, No. 1, 47–53, 7 figs. [Refs. in footnotes.]

The authors hold that patients with long-standing sickle cell anaemia have a fairly characteristic habitus which may be considered typical of the disease, and that awareness of this habitus may lead to the disease being suspected on inspection of the nude patient.

Such patients are slender and appear relatively tall, although they are not necessarily of greater than average height. The shoulders and hips are narrow, producing a linear type of habitus. In some the head is somewhat abnormal in shape, being dolichocephalic, scaphocephalic or otherwise unusual. The neck appears short. The normal upper dorsal kyphotic curvature is definitely accentuated and the lower lumbar curvature excessively lordotic. The arms and legs are thin and long, and the trunk is short. The chest is deep and narrow, producing a "hoop-chested" appearance. The hands and fingers, feet and toes are long and narrow; the hands have been well termed "spider hands." Signs of hypogonadism, such as genital hypoplasia or atrophy, hypotrichosis and high pitched voice, are encountered occasionally. In children the signs are either slightly or strongly abnormal, depending on the severity and duration of the disease, thin arms and legs, "hoop chest" and protruding abdomen being the most outstanding characteristics.

X-ray abnormalities are not constant. Changes in the skull are rare, and consist of decalcification with exaggeration of the normal reticulation, best seen in the vertex. The vertebral bodies sometimes show pronounced decalcification, and sometimes crescentic depressions of their superior and inferior borders, most noticeable in the lumbar region. The hands show "spider fingers" with pronounced decalcification in the metacarpal bones and phalanges in some cases.

Anthropometric measurements showed that the mean stature of the diseased patients was slightly less than that of control subjects, and that the mean weight of the patients was noticeably less. The span was less for patients than for the controls, but the relation of the span to stature was not significantly different in the two groups. Compared with stature the sitting height was shorter, while the average pubic height was greater in the patients than in normal persons. The average widths of the shoulders and pelvis were less in the diseased group than in the control, as were also the circumferences of the chest and abdomen. The antero-posterior diameter of the chest was relatively increased in the patients.

The anaemia is probably of great importance in altering the habitus, as patients with sicklaemia but no anaemia do not have an abnormal habitus.

The habitus of the normal adult American negro may be confused with that of those suffering from sickle anaemia. The normal negro is of short, light and slender build, the span exceeding the stature by as much as 12 cm. in some negro men. The habitus of patients with sickle cell anaemia also superficially resembles that encountered with primary hypogonadism. In sickle cell anaemia, however, the span does not exceed 107 per cent. of the stature, whereas in primary hypogonadism the ratio may exceed 112 per cent. In sickle cell anaemia the stature is often less than normal, while in primary hypogonadism it is greater than normal. The body weight in primary hypogonadism is not usually abnormally decreased, but in sickle cell anaemia it is usually sharply reduced. In both diseases the trunk is short compared with the stature, but in primary hypogonadism this is due to definite overgrowth of the long bones, whereas in sickle cell anaemia it is due mostly to decrease in height of the lumbar vertebrae. In sickle cell anaemia some of the adult patients with severe anaemia have evidence of gonadal hypofunction, but this is a secondary and not a primary type. *F. Murgatroyd.*

TOMLINSON, W. J. **Abdominal Crises in Uncomplicated Sickle Cell Anemia. A Clinico-Pathologic Study of 11 Cases with a suggested Explanation of their Cause.** *Amer. J. Med. Sci.* 1945, June, v. 209, No. 6, 722–41, 1 fig. [29 refs.]

With a view to throwing light on the causation of abdominal crises in sickle cell anaemia, a study was made of the clinical and autopsy findings in eleven cases showing this complication, which had been admitted to the Gorgas Hospital in the Panama Canal Zone.

Four of the patients were dead before they were seen by a physician. In the remaining seven the illness terminated fatally, and manifestations of shock were recorded in five. The clinical picture was characterized by sudden abdominal distress with rigidity, pain or tenderness, rigors, frequently jaundice, and peripheral vascular collapse. Severe anaemia was a constant feature, but since haemoglobinuria was absent, the anaemia was not thought to result from intravascular haemolysis.

The evidence of shock was found post mortem in all cases, the peripheral veins being collapsed and the internal organs showing marked congestion.

It is suggested that the mechanism of death in these cases is shock, and that the anoxia is aggravated because the sickled erythrocytes are ineffective carriers of oxygen and because they tend to stagnate in the small capillaries, thus increasing the circulatory failure and perpetuating the vicious circle of shock.

The desirability of treating such cases with blood or plasma transfusions is emphasized, but attention is drawn to the necessity of rejecting donors who show the sickling trait. *L. J. Davis.*

TRINCÃO, C. **The Sickle-Cell Trait in Saint Thomas Island.** *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 381–2.

TEIXEIRA, W. G. Hematias falciformes nos indigenas de Angola. [**Sickle Cells in the Natives of Angola.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 365–74, 2 figs. on 1 pl. English summary (9 lines).

SARMENTO, A. Contribuição para o estudo da anemia de células falciformes nos negros de Angola. [**Contribution to the Study of Sickle Cell Anaemia in the Negroes of Angola.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 345–50. English summary (6 lines).

VENOMS AND ANTIVENENES.

BOQUET, P. Sur la toxicité du sérum de *Vipera aspis.* [**The Toxicity of the Serum of** *Vipera aspis.*] *Ann. Inst. Pasteur.* 1944, Sept. Oct., v. 70, Nos. 9–10, 302–4.

PHISALIX and BERTRAND have stated that the serum of this viper is toxic by reason of the presence of the parotid gland venom in the blood. CALMETTE, ARTHUS and others have disputed this. The author has consequently estimated the toxicity of *V. aspis* venom and serum in *vivo* and their diastatic activity *in vitro.*

A minimum dose of 0·35 mgm. of the venom per kgm. body weight, intravenously injected kills a rabbit in 3–5 minutes, all the blood in heart and vessels being coagulated. For guineapigs of 450 gm. weight and the same venom similarly administered a dose of 0·1 mgm. is needed, and the blood in heart and vessels is fluid and not coagulable. If 1·5 mgm. are injected subcutaneously into a guineapig there is local oedema and sloughing, and death ensues in about 24 hours. Intraperitoneal injection of 0·05 mgm. into mice of 18–20 gm. kills in a few hours, the blood remaining fluid. Heating the venom to 60°C. reduces slightly its toxicity for the guineapig, but not for the other animals.

Fresh serum is much less toxic than the venom ; at least 6 cc. intravenously are needed to kill a rabbit of 2 kgm. in an hour, and the blood coagulates only slowly after death. When the serum has been heated to 56°C. for 30 minutes it loses its toxic properties. One cc. of fresh serum killed most guineapigs in less than an hour, but some lived longer. At autopsy the viscera were very congested and showed focal haemorrhages ; the blood is partly haemolysed and coagulates slowly ; subcutaneously administered 1 cc. causes in 24 hours local oedema followed by necrosis ; 0·1 cc. of the serum injected intraperitoneally into a mouse causes death in a few hours, but even five times this dose has no toxic effect if the serum is heated to 56°C.

In vitro the *venom* is found to haemolyse horse red cells by action of a lipoidase in presence of lecithin, and to coagulate the plasma ; heating to 60°C. does not affect these actions. The *serum* does not coagulate horse plasma, but haemolyses the red cells directly, without any need for added lecithin, and heating to 56°C. for 30 minutes inhibits this action.

If the serum contained venom as it is secreted by the glands, the toxic effect should be inhibited by antivenene, but it is not. *H. Harold Scott.*

DERMATOLOGY AND FUNGOUS DISEASES.

D'AVANZO, C. S. **Impetigo Bullosa in the Tropics.** *Arch. Dermat. & Syph.* 1945, July, v. 52, No. 1, 28–9.

" Impetigo bullosa is one of the more common dermatologic entities found among troops in the tropics, particularly in the infantry. The eruption appeared in 0·7 per cent. of all troops observed. Most patients with impetigo bullosa were treated in their units satisfactorily. They constituted 0·44 per cent. of all admissions and 3·2 per cent. of those hospitalized for cutaneous diseases. The most important factor involved in this disease is perspiration. Although it is less frequent, the incidence rate of impetigo bullosa parallels that of miliaria, because the same factors help produce both eruptions. Impetigo bullosa as found in the tropics should be removed from the classification of the impetigo

contagiosa group. It is a clinical entity, and sweat is the all-important factor.

"The most important part of treatment is prophylaxis. Proper personal hygiene is essential. The commanding officer of the unit has a definite function in prevention and much will depend on him particularly in infantry troops or other troops that do heavy manual labor. Clothing made of light porous material with a coarse weave is recommended for the use of troops in the tropics."

HARPER, A. R. **Pyodermia in the Tropics : with a Description of Three Cases.** *J. Roy. Nav. Med. Serv.* 1945, July, v. 31, No. 3, 170–72.

MCLAUGHLIN, R. R. M. **Treatment of Tropical Skin Diseases in the South Pacific: an Outline.** *U.S. Nav. Med. Bull.* 1945, Aug., v. 45, No. 2, 314–22.

The author records his opinions concerning some 5,000 dermatological cases seen during the course of medical duties with the U.S. Navy in the South Pacific. Of these cases 33 per cent. were suffering from fungous infections, 15 per cent. from pustular or cystic acne, 10 per cent. from ulcers of various types, 10 per cent. from dermatitis venenata (tropical woods or foliage) and about 6 per cent. from impetigo and ecthyma.

The main part of the communication consists of direct and sensible instruction concerning the treatment of the commoner cutaneous diseases, but there are two points which attract the abstracter's attention. The first is the recommendation to treat pediculosis pubis by spraying the infested area with Freon Aerosol Insecticide ; the second is the author's insistence that, contrary to popular belief, pustular and cystic forms of acne vulgaris are aggravated by tropical service and that this eruption may develop in hot climates in men who have not previously suffered from the malady.

[Without doubt the author is correct in his thesis that, under conditions of active service in hot and—one may add—humid, climates, acne vulgaris may be severely aggravated. He may also be quite correct in his assertion that the eruption may develop *de novo* in adult males working in such areas ; some would maintain that any adult over, say, 25 years of age who suddenly develops severe acne in the tropics probably suffered from the eruption in a sub-clinical form in adolescence.] *R. M. B. McKenna.*

SOBERÓN Y PARRA, G. & LEÓN Y BLANCO, F. Las moscas del género *Hippelates* como posibles vectores del mal del pinto (2a. comunicacion). [**Flies of the Genus Musca as possible Vectors of Pinta.**] *Ciencia.* Mexico. 1944, v. 4, No. 11/12, 299–300. [Summary taken from *Rev. Applied Entom.* Ser. B. 1945, Aug., v. 33, Pt. 8, 125.]

The authors refer to experiments in Mexico described in a paper that has not been received at this Institute (F. LEÓN Y BLANCO & G. SOBERÓN Y PARRA. Nota sobre la transmisión experimental del mal del pinto por medio de una mosca del género *Hippelates. Gac. méd. Méx.* **71,** pp. 534–539, 1941) in which they showed that the disease known as mal del pinto [or carate] could be transmitted by flies of the genus *Hippelates.* An account is given in the present paper of investigations on the persistence of the spirochaetes [*Spirochaeta caratea*] in flies of this genus that had fed on serum from an opened lesion on an infected person. Mobile spirochaetes were present in all cases in serum from the digestive tract of the flies 10–30 minutes after an infective meal, spirochaetes were in all cases present but some had lost their mobility 40–70 minutes after the meal, and fewer could be seen and most were motionless after 80–120 minutes. Only very occasional motionless spirochaetes were seen when more than two hours had elapsed. There were spirochaetes in three out of 11 specimens of vomit drops, each from 4–10 flies, collected 15–20

minutes after an infective meal and examined immediately. No spirochaetes were found in the contents of the digestive tract of 31 adult *Hippelates* taken in nature.

DI PIETRO, A. Granuloma nasal a *Rhinosporidium seeberi* (8a observación argentina). [**Nasal Granuloma due to** *R. seeberi*.] *Rev. Asoc. Med. Argentina.* 1945, June 30, v. 59, No. 560, 704–7, 6 figs.

SULZBERGER, M. B., SHAW, H. C. & KANOF, A. **Evaluation of Measures for Use against Common Fungous Infections of Skin; Screening Tests by means of Paired Comparisons on Human Subjects.** *U.S. Nav. Med. Bull.* 1945, Aug., v. 45, No. 2, 237–48.

In this paper the authors record the result of an investigation made amongst a controlled population in a Naval Disciplinary Barracks to determine the efficacy of five different medicaments in the prophylaxis of fungous infections of the feet and groin. In all, 808 volunteers were used for the experiment, which was made during a period of 3–4 months in 1944.

The materials investigated were :—

(*a*) Undecylenic acid–zinc–undecylenate powder.
(*b*) Sodium propionate powder.
(*c*) Boric acid–salicylic acid powder.
(*d*) Undecylenic acid–zinc–undecylenate ointment.
(*e*) Clothing impregnated with Impregnite CC3 by an oil-in-water emulsion process. The formula of CC3 is not stated; the formulae of the powders and ointment were as follows :—

Propionate powder (pigmented)

	Per cent.
Sodium propionate (du Pont " Mycoban ")	20·0
Talc, U.S.P.	79·5
Polychloro copper phthalocyanine (green)	0·5

Undecylenic acid–undecylenate powder (pigmented)

	Per cent.
Zinc undecylenate	20·0
Talc, U.S.P.	76·0
Undecylenic acid, Grade AA	2·0
Dibenzo thio indigo (red)	2·0

Boric acid–salicylic acid powder (pigmented)

	Per cent.
Salicylic acid, U.S.P., powder	2·0
Boric acid, U.S.P., impalpable powder	6·0
Zinc stearate, U.S.P.	3·0
Talc, U.S.P.	86·5
Dibenzo thio indigo (red)	2·0
Polychloro phthalocyanine (green)	0·5

Undecylenic acid–undecylenate ointment (pigmented).

	Per cent.
Undecylenic acid, Grade AA	5·0
Triethanolamine	3·0
Zinc undecylenate	18·0
Propylene glycol, N.F.	10·0
Carbowax, 1,500	19·0
Carbowax, 4,000	29·6
Water, distilled	15·0
Dibenzo thio indigo (red)	0·4

Experiments in prophylaxis were limited to the protection of the feet from fungous infections ; 567 men who were free from active infection of this type were divided into five groups. One group was used as a control and did not receive treatment ; four groups were treated as follows :—

Group A.—Undecylenic acid undecylenate powder for the right foot ; boric and salicylic acid powder for the left foot.

Group B.—Sodium propionate powder on the left foot, boric and salicylic acid powder on the right foot.

Group C.—Undecylenic acid–undecylenate powder on the right foot ; sodium propionate powder on the left foot.

Group D.—A sock impregnated with CC3 on the right foot ; no treatment to the left foot.

In the control group, 8·85 per cent. of the unprotected feet developed fungous infection. The percentage ratios of feet which became infected during the period in which the various prophylactics were used were as follows :—

1·07 per cent. treated with undecylenic acid–undecylenate powder ;
3·10 per cent. treated with sodium propionate powder ;
8·33 per cent. clothed with socks impregnated with CC3 ;
11·15 per cent. protected with boric and salicylic powder.

Therefore in this investigation the undecylenic acid and the sodium propionate powders gave the best protection, whilst the use of impregnated socks, or boric and salicylic acid powder appeared not to confer benefit to the users.

With regard to the apparent advantages of the use of the undecylenic acid preparations and the propionate powder the authors are not prepared to say more than that they appear to afford some degree of protection against fungous infections of the feet, the former powder being perhaps somewhat superior to the latter.

When the preparations were used for the treatment of active fungous infections of the feet (160 persons) and groin (72 persons) undecylenic acid–undecylenate powder and sodium propionate powder were found to be effective on both areas, the former being more effective than the latter, particularly in the crural area. Undecylenic acid–zinc undecylenate applied to the feet in an ointment vehicle was not superior to the same ingredients incorporated in a powder. Shorts and socks impregnated with CC3 did not produce any significant therapeutic effect ; the shorts produced irritation of the skin in about one-third of 11 patients who wore them.

The authors found that in six cases of pityriasis versicolor the use of undecylenic acid–zinc undecylenate ointment and propionate acid–sodium propionate ointment failed to produce any therapeutic response ; they suggest that this may be associated with the preference shown by the causal fungus (*Microsporon furfur*) for cutaneous areas where the sweat is more acid, for this preference may indicate that the fungus has an inherent resistance to fatty acids and their salts.

[Besides the findings stated, the communication has many points of interest. The authors are extremely conservative ; they claim nothing more than that the results are sufficiently encouraging to warrant field trials of the better preparations. Their technique of using paired comparisons on human subjects is of interest, and the device of giving each preparation a distinctive colour is an ingenious method of circumventing human fallibility. They emphasize that for practical purposes powders are preferable to ointments.

The investigation seems to have been well controlled. It is interesting to note that, although numerous patch-tests of the ingredients used failed to produce any cutaneous reactions (although in many instances the tests were repeated twice with an interval of four weeks between them), impregnated

shorts caused irritation in about one-third of the wearers. It has been found in Britain, in the investigation of cases of textile dermatitis, that patch tests made with the suspected material frequently fail to produce any reaction in the skin, whilst wearing of the suspected garment may rapidly produce a cutaneous inflammation. Also it may be noted that, in infections of the groin, the undecylenic acid powder and the propionate powder required an average period of 3·5 and 5·5 weeks respectively to produce a cure; this is a slow rate of therapy. The time factor was more satisfactory with regard to the feet, cure being obtained with both preparations in 4·6 weeks.

This communication may be integrated with the papers by KEENEY, AJELLO and LANKFORD published in the *Bull. Johns Hopkins Hosp.*, 1944, v. 75, pp. 377, 393 and 410]. *R. M. B. MacKenna.*

TROPICAL OPHTHALMOLOGY.

A REVIEW OF RECENT ARTICLES. XLVI.*

Conjunctivitis.—The treatment of acute conjunctivitis and trachoma with sulphonamides is discussed by MITERSTEIN and STERN.[1] During the 1941 summer epidemic of Koch-Weeks conjunctivitis, they examined the use of sulphanilamide and some of its derivatives in the local and oral treatment of this disease. Koch-Weeks conjunctivitis in the Near East appears in epidemic form in the summer and early autumn and affects children especially. It is highly contagious, usually attacks both eyes and is characterized by a profuse discharge with much swelling of the eyelids and conjunctiva. The authors started their experiments with " Pyranil " (Teva Ltd.) which had shown strong bactericidal activity *in vitro* and *in vivo*. Pyranil is a compound containing *p*-aminophenyl-sulphonamide [sulphanilamide] in addition to pyridine-dicarbonic acid anhydride. An ointment was prepared which contained 5 per cent. of the active principle in an adequate ointment base. In 13 cases of Koch-Weeks conjunctivitis, one eye was treated with Pyranil ointment and the other with 2 per cent. silver nitrate. The results in 12 cases were equally good in both eyes. In a few more cases of Koch-Weeks conjunctivitis, both eyes were treated with Pyranil ointment as well as with silver nitrate. The combination gave excellent results and encouraged them to try sulphanilamide and sulphapyridine by the mouth. The former had no effect, but the latter, given by the mouth without any local treatment, cleared up the conjunctivitis completely in so short a time that treatment with silver nitrate seemed slow in comparison. About 50 cases of all ages were treated and the following course gave the best results: A total of 0·05 gm. per kgm. body weight per day is given in divided doses. The intervals must not be too long, every 2–3 hours seems to be the best interval. On the 2nd and 3rd day this dosage is repeated; during the next 4–5 days, it is gradually diminished. If slight swelling and injection of the conjunctiva remains, treatment is to be continued with argyrol, noviform ointment or some other antiseptic ointment. In the treatment of trachoma, including the corneal complications, the authors obtained excellent results with pyranil ointment, and in severe cases resisting every therapeutic attempt, this ointment is the treatment of choice though it does not exclude the use of atropine and sulphanilamide by mouth or make it unnecessary to squeeze out trachomatous granules if they need it.

---

*For the 45th of this Series see Vol. 42, pp. 486–490.

[1]MITERSTEIN, B. & STERN, H. J. Treatment of Acute Conjunctivitis and Trachoma with Sulphonamides. *Lancet.* 1945, May 26, 649–50, 3 figs.

The effect of various dosage with sulphapyridine, sulphathiazole, sulphadiazine and penicillin in the treatment of the acute epidemic ophthalmias (Koch-Weeks and gonococcal) of Egypt was studied by BLAND and WILSON.[2] They found that a single dose of a sulphonamide will cause a reduction of bacteria in four hours and total suppression of bacteria in about 12 hours. A considerable proportion of gonococcal cases will be cured by a single dose, but Koch-Weeks cases are more resistant. Two doses on two successive days (3 doses in all) will cure all cases of gonococcal and Koch-Weeks ophthalmia, with very rare exceptions. A single intramuscular dose of penicillin will reduce gonococci to *nil* in 3–4 hours, but repeated doses at short intervals are necessary to ensure cure. Relapses occur in 10–12 hours after a single dose. Penicillin has no effect on Koch-Weeks cases and is, therefore, unsuitable as a treatment for acute Egyptian ophthalmia.

Those who know the conditions of life in Egypt and other oriental countries, where similar conditions of life and disease prevail, will realize the importance of these findings. Acute ophthalmia is the cause of 80 per cent. of the blindness (total blindness 1 per cent., monocular 4 per cent.) of Egypt. The increased ocular secretion caused by the disease promotes also the spread of trachoma, that second universal plague of Egypt, itself a fertile source of damaged sight. The authors point out that no treatment which requires hospitalization or even attendance at hospital can hope to do more than scratch the surface of this problem of eye disease unless treatment can be taken to the villages, where the great majority of the people live, and there simply and easily administered. They believe the treatment with sulphonamides provides the necessary solution as it is simple, safe, effective and could be given by unqualified persons with a minimum of training.

GUTMANN[3], writing on allergic warm season conjunctivitis, considers that conjunctivitis vernalis (spring catarrh or vernal conjunctivitis) as seen in Palestine is badly named in that the name is not sufficiently comprehensive; it would be more appropriately called an "allergic warm season conjunctivitis" (a.w.s.c.). The experiments he reports are based on 180 cases, 36 of whom were examined and treated by the author over the last eight years. An analysis of the age of patients showed 10·5 per cent. have been between 1–5 years, 24 per cent. between 5–10 years, 21 per cent. 10–15 years, 7·5 per cent. between 15–20 years, 28·5 per cent. between 20–30 years, and 7·5 per cent. 30 years and more. He found that the disease is three times as common in males as in females, and that girls very seldom contract it. He considers that hormonal factors may be of importance, and that the combination of heat and moisture are the climatic factors responsible for the condition.

Allergic warm season conjunctivitis (spring catarrh) has nothing to do in any way with hay fever, although in patients with a.w.s.c. he found allergic reactions to bacterial allergens and moulds (fungi). The average of the blood eosinophiles was 9·6 per cent., there were numerous eosinophilic cells in the lachrymal secretion; the blood sedimentation rate and the blood calcium level were normal. More than half the patients had suffered previously from other allergic diseases, and hereditary cases were often seen. The allergic skin reaction to moulds is similar to that for bacterial allergens, namely the late reaction; but this is not exclusively so and the patients may show the "nodular, typical lagging reaction." The a.w.s.c. appears as a secondary allergic disease, the primary cause of which is to be sought in some sort of chronic infection such as

---

[2]BLAND, J. O. W. & WILSON, R. P. Bacteriological and Clinical Observations on the Treatment of the Acute Ophthalmias of Egypt with Sulphonamides and Penicillin. *Brit. J. Ophthalmology.* 1945, July, v. 29, No. 7, 339–55. [10 refs.]

[3]GUTMANN, M. J. Allergic Warm Season Conjunctivitis (Conjunctivitis Vernalis, Spring Catarrh, Vernal Conjunctivitis) as an Allergic Disease. *Acta Med. Orientalia* (*Palestine & Near East Med. J.*) 1945, May, v. 4, No. 5, 150–55. [16 refs.]

infected teeth, tonsils, sinuses, etc., upon which a fungal allergy is superimposed. The fungi most frequently found were: *Alternaria, Hormodendron* and *Cladosporium, Penicillium, Aspergillus, Monilia,* and yeasts.

Treatment consists in the removal of foci of infection, and in reorganization of the patient's surroundings. For desensitization a mixed extract of various moulds or from the material of the patient's surroundings, fortified by bacterial allergens, is used. All injections are given intracutaneously for at least six months and in most cases for 1–2 years.

Trachoma.—Mass education, school medical services, and defective vision, with special reference to trachoma in East Africa, are discussed by Howard Reed.[1] The pamphlet entitled "Mass Education in African Society" (Colonial No. 186) which has been published by the Advisory Committee on Education in the Colonies, is of great interest to all concerned with post-war development in our African colonies. Two main lines of attack are suggested—the provision of schools so that children may receive the rudiments of learning, and the fostering of voluntary groups with the provision of literature in the native language, so that young literate people may be helped to continue their education after leaving school. The most effective way of assisting mass education is to concentrate upon cultivating a state of "positive health" in schoolchildren by developing an efficient school medical service, in which the care of eyesight plays a very important part. Inability to see clearly prevents accurate observation, and eye-strain caused by visual defects induces a distaste for learning. Medical inspection and treatment of schoolchildren should provide for routine eye examinations. External diseases of the eyes such as conjunctivitis and corneal ulceration are prevalent in the tropics and cause much defective vision. Of these, trachoma is far the most important, and its aetiology still remains unknown. The present incidence of trachoma in the East African colonies is very difficult to estimate with any degree of accuracy, but there is no doubt that it is more widespread than is generally realized. According to Jones the percentage of trachoma cases in Uganda schools is 13, and according to Boase "it is very prevalent in Uganda." Harley-Mason gives the percentage of trachoma cases attending the Nairobi Eye Clinic for 1941–43 as 12 to 17 per cent. In Mwanza the author found that in 232 native eye cases, 21·5 per cent. were undisputable cases of trachoma. Wilson working in Masailand says the disease is common. McDonald found in Lindi that trachoma was the chief cause of rejection of recruits. Out of 4,000 men examined in 1943, there were 23·3 per cent. rejected for trachoma. In Egypt, where trachoma is so widespread, MacCallan found that very heavy infection occurred in pockets. An irregular distribution has also been found in India, and it is probable that when a survey is made in East Africa, the degree of trachoma infection will vary greatly in different areas. It would appear that the trachoma at present seen in East Africa is of a less virulent type than that seen in Egypt and India.

The author issues a warning that a post-war increase of trachoma in East Africa may follow the return of the East African troops. This would be due to the large scale movements of African troops and labour corps to the Middle East and Far East, in which areas the incidence of trachoma is high, and would necessarily cause a corresponding adverse effect upon the execution of the plans for mass education. The following methods might be adopted to estimate the danger of an increase of trachoma after the war:—

1. The present incidence of trachoma in East Africa should be estimated; this could most accurately be done by the examination of the eyes of schoolchildren in given areas.

[1] Reed, H. Mass Education, School Medical Services and Defective Vision, with special reference to Trachoma in East Africa. *East African Med. J.* 1945, Feb., v. 22, No. 2, 46–55.

2. The Army Medical Services should be requested to make a special investigation, and to supply statistics of the degree of infection among East African troops. This should be carried out by a medical officer having a special knowledge of trachoma.

To combat trachoma, the following measures might be introduced :—

1. Regular medical inspection of schools, including village schools. A circular giving an account of the disease and its treatment could usefully be issued to all medical officers.

2. Children are usually infected during the first two years of life, so that early cases could be reached to some extent by giving instruction in the diagnosis of the disease to health visitors and those connected with child welfare and maternity clinics. In these clinics, infected mothers could be discovered, treated and rendered less infectious when the baby is born.

3. The daily administration of eye-drops to infected schoolchildren by the teachers until such time as a school medical service is introduced.

4. Special eye clinics should be opened in central towns for treatment as well as for teaching the subordinate medical staff.

5. Africans in the military forces should receive adequate treatment before demobilization, to render them non-infectious.

The surgical treatment of trachoma in Uganda is described by BOASE[5]. In a recent sample survey of 504 blind persons in a part of the kingdom of Buganda, he found that trachoma was responsible for blindness in 30 per cent. of these cases. It is found in all sections of the community, in all stages ; it varies in its manifestations from a type so severe as to ridicule text-book descriptions, to a type so mild that only a fortuitous examination will disclose its presence. The incidence is markedly affected by standards of hygiene and nutrition. Treatment on classical lines is tedious, painful and prolonged, and few persevere to the stage of arrest or cure. The author's method (a surgical one) holds a prospect of speedier relief in cases of severe trachoma in stage 2 and in all cases showing trichiasis or entropion. The operation consists of a sub-total resection of the upper tarsal plate (and sometimes the lower). There is nothing original in this procedure ; it is sometimes known as Heisrath's operation and is described by MacCallan under the somewhat cumbersome title of " combined excision of tarsus and conjunctiva." The descriptive term " tarsectomy " is widely used by other writers and has the advantage of brevity. The following description applies to the author's technique :— Surface anaesthesia of the eyeball is obtained with drops of 1 per cent. Pantocaine (or Decicaine). The lid is infiltrated with 2 cc. of 2 per cent. procaine-adrenaline solution. The needle is introduced in the centre of the lid and 1 cc. injected towards each canthus. Adequate eversion of the lid is essential for facile performance of the operation. This is best fulfilled by Cruickshank's entropion forceps (Down's catalogue 724/7), which is applied solely to the skin of the lid, the spatula blade is placed in the hollow between the globe and the supraorbital margin and the 3-pronged blade on the lid 2 to 3 mm. above the lashes. The forceps is then closed and secured with the fixing screw, thus firmly holding a sizeable fold of skin, subcutaneous tissue and orbicularis muscle between the blades. When the handle is depressed towards the forehead, the lid is automatically and satisfactorily everted. It is then clipped to the towel with a towel clip to prevent it from flying back towards the cheek. A sharp knife is used to divide the tarsal plate along its whole length at about the level of the subtarsal sulcus. The incision is commenced at the inner canthus for the right eye and at the outer canthus for the left eye and is

[5]BOASE, A. J. The Surgical Treatment of Trachoma. *East African Med. J.* 1945, Feb., v. 22, No. 2, 56–61.

extended across the lid, keeping about 1½ mm. from the margin. The section must be made with small sawing movements, as the average trachomatous tarsus is very dense. It is of paramount importance to keep the line of the incision at a uniform distance from the lid margin. The tarsus is now seized with a fixation forceps or a small vulsellum at the centre of the proximal lip of the incision, and with a few light strokes of the knife is separated from the overlying tissue. The dissection is continued upwards for 3 to 4 mm. above the upper edge of the tarsal plate so as to mobilize the conjunctiva in this area. This is more conveniently done with scissors. The next step is to cut through the conjunctiva as close as possible to this upper edge. Haemorrhage may be a nuisance but is easily controlled by twisting off the severed vessels. Three 8-inches long, double-armed cotton thread sutures on No. 3 or 4 half curved needles are required. The cut edge of the conjunctiva is seized at its centre and pulled down. One needle of the double-arm suture is passed through the conjunctiva from its superficial aspect close to the cut edge and then through the lid just above the stump of the tarsus so that it emerges on the skin close to the middle prong of the eversion forceps, that is 2–3 mm. above the lashes. The second needle of this suture is then passed through the stump of the tarsus and out through the skin 1 mm. or so nearer the lashes than the first needle. The remaining two double-arm sutures are inserted in a similar manner on either side of the central one, and at points half way between it and the canthi. The eversion forceps is now removed and the lid allowed to fall into place. The sutures are pulled taught, and after the cut edge of the conjunctiva has been adjusted to that of the tarsal stump, they are tied over a thin strip of rubber dam which facilitates their subsequent removal. With practice the operation can be performed on both eyes in the space of 15 to 20 minutes.

[The reviewer, with a large experience of trachoma, agrees with MacCallan that the operation of tarsectomy is unsuitable for the relief of simple trichiasis and should be reserved for cases of trachoma stage 2 where there is gross, shaggy, papillary hypertrophy of the conjunctiva. Webster's mucous graft operation is a very satisfactory one for the varying degrees of trichiasis-entropion.]

The aetiology of trachoma is critically reviewed by BLAND[6] and in the course of this review he points out that the four cardinal diagnostic signs of trachoma in man are: The presence of inclusion bodies; the formation of follicles on the tarsus; vascularization of the cornea; and scarring of the conjunctiva. The available evidence shows that trachoma is a specific infectious disease caused by a non-cultivable, filterable agent, identical with the elementary and initial bodies found in the Prowazek-Halberstaedter inclusions, and bearing a close natural relationship to the viruses of inclusion conjunctivitis, lymphogranuloma inguinale and psittacosis. There is no evidence that this agent possesses an arthropod host, nor that the louse is a vector of the disease, though the agent may survive for some days in the body of the louse. Are we to consider the trachoma agent, along with the three other agents to which it is related, as rickettsiae or viruses? Since at present there is not complete agreement among bacteriologists as to the precise definition of the genus *Rickettsia*, and since the virus group is largely defined by negative characteristics and possibly contains agents of very different intrinsic nature which a natural biological classification would not place together, the matter becomes largely one of personal choice. Undoubtedly those four agents resemble the rickettsiae in a number of points; they are minute in size and show pleomorphism, though not the bacillary and thread forms of typical

[6] BLAND, J. O. W. The Aetiology of Trachoma. A Critical Review of Present Knowledge. *Brit. J. Ophthalm.* 1945, Aug., v. 29, No. 8, 407–20. [58 refs.]

rickettsiae ; they do not stain with aniline dyes ; they are Gram-negative ; they stain easily with Giemsa and in their various morphological forms take a variety of tints from red to dark blue with this stain ; they stain easily with Castañeda's stain ; they are only little, if any, more filterable than rickettsiae ; they cannot be cultivated apart from living cells and they adopt an intracellular habitat. Non-cultivability, intracellular habitat, lack of staining with aniline dyes and Gram-negativity are points which are shared both by viruses and rickettsiae. These four agents including trachoma differ from the typical large virus : (1) In possessing initial bodies which stain blue with Giemsa and in showing greater pleomorphism ; (2) in forming inclusions with a basophilic matrix ; (3) in staining with Castañeda's stain. They differ from typical rickettsiae : (1) In lack of an arthropod host ; (2) in not showing bacillary and thread forms ; (3) in forming inclusions with a matrix.

The author himself is of opinion that the four agents stand in an intermediate position between the rickettsiae and the large viruses and may possibly form a biological link between them. For the present, he prefers to group them with the viruses but to give them a distinctive position as the " basophilic viruses " on account of the blue staining of their initial bodies and of the matrix of their inclusions, which distinguishes them from the larger typical viruses which do not possess blue initial bodies and whose inclusions are acidophilic.

Keratitis.—CHAMBERLAIN and BRONSON[7], working amongst troops exposed to malaria, have found an increased incidence of keratitis associated with herpes simplex in a population heavily infected with malaria. Amongst new patients attending the ophthalmological clinic over a period of 18 months, there were more than six times the number from a malarial division as from one that had not been exposed to malarial infection. The incidence, however, is very low, less than 0·2 per cent. of patients with acute malaria became affected with herpetic keratitis. Early diagnosis and intensive treatment of malaria are essential in the management of herpetic keratitis occurring in patients with malaria. Recurrent attacks of malaria are often associated with a reactivation or an extension of the corneal ulceration.

Sulphadiazine powder, applied locally, seems to promote rapid healing, and the authors suggest that it may prove a satisfactory adjunct to the accepted treatment of dendritic keratitis with strong solution of iodine.

Papillitis.—Malarial papillitis amongst the troops in the New Guinea campaign is discussed by LEWY[8]. Although this ocular complication has hitherto been scantily described, he considers that it is a common concomitant of recurrent malaria and should be regarded as a papillitis rather than papilloedema because of the injected appearance of the nerve head in the majority of the patients, diminution in visual acuity, reduced peripheral fields, and enlarged blind spots. It is improbable that the condition is a result of medication as none of the patients presented the retinal ischaemia, arterial spasm and blindness characteristic of quinine amblyopia, which is often the cause of toxic retrobulbar neuritis resulting in complete central blindness and pallor of the disc. There appears to be no direct correlation between the degree of oedema and inflammation of the optic disc and the size of the blind spot, neither could any absolute correlation be made between the amount of papillary change and the number of attacks of malaria.

---

[7]CHAMBERLAIN, W. P., Jr. & BRONSON, L. H., Jr. Herpes Simplex Keratitis in Malaria : Clinical and Experimental Study. *Arch. Ophthalmology.* 1945, Mar., v. 33, No. 3, 177–83.

[8]LEWY, R. B. Malarial Papillitis. *War Medicine.* Chicago. 1945, June, v. 7, No. 6, 341–4, 3 charts.

Penicillin.—The practical uses of penicillin in the everyday practice of ophthalmology are discussed by HARDESTY[9]. In surface infections, such as conjunctivitis and corneal ulcers, local rather than systemic use was employed, with good results, in strengths of 20,000 units per cc. of isotonic salt solution. Two or three drops were instilled into the conjunctival sac every half hour and then every hour. Within 24 hours considerable improvement was noted, and within 10 days the eyes were quiet. In cases of non-traumatic orbital cellulitis, non-traumatic uveitis and non-luetic keratitis, systemic use of penicillin together with appropriate local treatment has proved decidedly beneficial. The author quotes Stokes, Sternberg *et al.* as having reported improvement in some cases of luetic interstitial keratitis, but in the treatment of luetic optic neuritis, penicillin should be given with great care as frequent Herxheimer reactions are reported from its use. He also quotes Sievers, Knott and Soloway as having reported good results from intramuscular injections of penicillin in gonorrhoeal ophthalmia. In endophthalmitis following penetrating wounds, and post-operative intra-ocular infection, he advocates daily subconjunctival injections of 0·25 cc. of isotonic salt solution containing 2,500 units of penicillin, together with full dosage of the drug intramuscularly. He concludes by saying that with penicillin the practice of ophthalmology has an implement to combat successfully superficial and deep ocular diseases that have heretofore put up a stubborn and unyielding front to all other treatments.

Riboflavin deficiency as a cause of eye disease.—Vitamin B_2 deficiency as a cause of eye diseases in Bengal is discussed by KIRWAN, SEN and BOSE[10]. In India cases have been reported from Madras, but as a result of their investigations the authors found that the incidence of this deficiency in Bengal is low and the occurrence of ocular complications due to riboflavin deficiency is rare. Angular stomatitis, burning sensation in the mouth while taking food, fissuring of the tongue, seborrhoeic dermatitis of the face with multiple comedones, itching and scaliness of the skin of the scrotum are the main general signs and symptoms.

The ocular manifestations of "ariboflavinosis" varied from an itching, burning sensation of the eyes to mild or even severe photophobia accompanied by dimness in inadequate light, and even to partial blindness. Injection of the palpebral conjunctiva, circumcorneal injection with proliferation of the limbic blood vessels, and superficial keratitis were seen in some cases.

The authors are of the opinion that patients with these signs and symptoms are suffering from a deficiency of more than one vitamin.

Blindness and diseases of the eye in South Africa.—In an article on this subject BOSHOFF[11] points out that there is in existence a very good system for the registration of blind Europeans, and that before they become registered for a pension, blind persons must be certified by an ophthalmologist. Hence the causes of blindness are known, but these European statistics are not as complete as they should be, as self-supporting and non-pensionable blind persons are not on a register of any kind. A Government report (1936) reveals that per 100,000 Europeans in 1908, 1911 and 1936 there were 81·24, 76·47 and 81·06 blind perons respectively.

Amongst non-Europeans the statistics are unreliable and are an understatement of the true facts, as non-Europeans can be certified by a district

---

[9]HARDESTY, J. F. The Use of Penicillin in certain Eye Diseases. *Southern Med. J.* 1945, June, v. 38, No. 6, 411–13.

[10]KIRWAN, E. O'G., SEN, K. & BOSE, N. Vitamin B^2 Deficiency as a Cause of Eye Diseases in Bengal. *Indian J. Med. Res.* 1944, Oct., v. 32, No. 2, 109–15. [33 refs.]

[11]BOSHOFF, P. H. Blindness and Diseases of the Eye in South Africa. *South African Med. J.* 1945, May 12, v. 19, No. 9, 148–9.

surgeon. Those placed on the register are only those who actually apply for registration and are fortunate enough to persuade the local native commissioner to grant such a pension ; if the blind person's family can support him a pension is not granted. Many persons with curable blindness (such as cataract) fail to seek rehabilitation by surgical means and become a permanent State liability.

For coloured persons there is an incidence of 127·74 blind per 100,000 sighted and partially sighted persons. Amongst the Bantu the incidence of blindness works out to be 400 blind per 100,000 sighted and partially sighted persons. These high figures are upsetting, but it is even more alarming to know that in certain districts, *i.e.* North Western Cape, Western and Northern Transvaal, the incidence varies from 1,000 to 2,000 blind per 100,000 sighted and partially sighted natives. In Mafeking, Zeerust, Marico and Rustenburg districts the incidence is 1,800 blind per 100,000. The author points out that money would be better spent on preventive work than on providing pensions for the blind. As a result the present expenditure on the blind would be gradually reduced and progress would be discernible. At present not a penny of the pension money is usefully employed. In his opinion the problem of blindness and eye disease in South Africa is desperately bad ; urgent and efficient action is needed. He advocates more interest by Government as to its responsibility in the matter and recommends intensive post-graduate training in ophthalmology in the medical schools.

Eye Diseases in the East are discussed by MINTON[12] in a brief article. He points out the prevalence of epidemics of conjunctivitis in the Middle East every year, in the spring and late autumn. They are of a highly virulent nature and often involve the cornea. They are due to Koch-Weeks bacilli, gonococci, diphtheria bacilli and staphylococci. He advocates general treatment with sulphapyridine or sulphathiazole, and local treatment with 30 per cent. sulphacetamide drops. He also discusses the prevalence of trachoma and its treatment with the sulphonamides, the presence of vitamin A deficiency in India and Ceylon, epidemic kerato-conjunctivitis during the summer in India. He ends by saying that the incidence of eye infection amongst Europeans living in the tropics is low, and that the tropical sun is not harmful to the eyes and is not responsible for eye diseases. *E. O'G. Kirwan.*

TROPICAL ULCER.

PRICE, E. W. **Spinal Anaesthesia in the Treatment of Tropical Ulcers.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1945, Sept., v. 39, No. 1, 83–6.

Chronic ulcers of the leg, as encountered in medical practice in the tropics, occasionally resist all the usual methods of treatment ; two such cases in which cure rapidly followed therapeutic spinal anaesthesia are described in this paper.

The first case was in an African boy, aged 14 years, who had ulcers on both feet spreading on to the dorsal and plantar aspects from toes infested with jiggers. He was treated in hospital for six weeks without being cured and was then given an intraspinal injection, between the 3rd and 4th lumbar vertebrae, of 75 mgm. of procaine in 1 cc. of sterile water and was immediately turned on to his back with his knees drawn up for one minute. Five days afterwards the ulcers were completely healed.

The second case was in an adult leper, aged 25, who had foul-smelling ulcers on the sole of his left foot. After treatment in hospital for 10 days without effect, he was given 150 mgm. of procaine in 1·5 cc. of water intraspinally, and six days later all the ulcers had healed.

---

[12]MINTON, J. Eye Diseases in the East. *Practitioner.* 1945, Sept., v. 155, No. 3, 176–9.

The author suggests that prolonged arteriolar spasm in the ulcerated area, rather than the presence of endarteritis, causes chronicity of the ulcer. The action of the anaesthetic may be a breaking of a reflex rather than a maintenance of vasodilatation. OCHSNER (*Surgery*, 1939, v. 5, 491) found that paravertebral block with novocain [procaine] immediately relieved the clinical symptoms of thrombophlebitis and the relief was maintained. *J. F. Corson.*

HAMMOND, A. G. **Occlusive Dressing—the Almaza Patch.** *East African Med. J.* 1945, July, v. 22, No. 7, 241–2.

An adhesive plaster of Paris dressing, the "Almaza Patch," was successful in the treatment of sores and ulcers under field conditions in a sandy area. Plaster of Paris three parts, tragacanth one part and acriflavine powder 0·001 part were mixed by grinding in a mortar. A piece of open-wove bandage, a little larger than the sore to be dressed, was cut and the powder thoroughly rubbed into its meshes in a mortar. A little of the powder was sprinkled on the sore, then the patch was removed from the mortar with forceps, immersed in eusol or water, applied to the sore, and carefully moulded with a palette knife; the patch was kept at rest until dry and set.

The dressing was left until the sore healed, when it dropped off or became loose. Indolent ulcers were treated with silver nitrate before the patch was applied.

The dressing was comfortable, did not restrict movement, and did not irritate the skin; it was very economical, as an ordinary 50-cigarette tin held enough powder for 500–600 dressings. *J. F. Corson.*

MISCELLANEOUS DISEASES.

GROSFILEZ & LEFÈVRE. Les maladies transmissibles observées dans les colonies françaises et territoires sous mandat pendant l'année 1938. [**Transmissible Diseases in the French Colonies and Mandated Territories during 1938.**] *Ann. de Méd. et de Pharm. Colon.* 1940, Apr.–May–June, v. 38, No. 2, 183–359.

DICK, G. W. A. **Routine Stool Examination.** *East African Med. J.* 1945, July, v. 22, No. 7, 237–40.

Microscopical examination of the faeces of British, African and Italian troops at an East African Casualty Clearing Station showed the following proportions (percentages) of carriers of helminths and protozoa:—

Parasite	African (3,000)	British (400)	Italian (400)
	Per cent.	Per cent.	Per cent.
Schistosoma mansoni ...	1·4	0·25	—
Taenia saginata	5·3	1	2·5
Ancylostoma	4·4	0·25	0·25
Strongyloides stercoralis ...	5·8	2·3	2
Enterobius vermicularis ...	0·1	0·25	0·25
Ascaris lumbricoides ...	2·8	—	0·8
Entamoeba histolytica ...	9·1	11·3	10·3
Entamoeba coli	9·8	7·5	12·5
Chilomastix mesnili... ...	8·2	11	12·5
Trichomonas hominis ...	6·4	5·3	6·8
Giardia intestinalis... ...	0·9	2	3
Iodamoeba bütschlii ...	1·6	0·25	1
Endolimax nana	1·6	—	—
Tricercomonas intestinalis ...	0·2	—	—

The figures of the Africans suggested that the medical treatment of helminthiasis in the East African Command had been satisfactorily carried out.

A cellular exudate characteristic of acute bacillary dysentery was found in 18·7 per cent. of Africans, 16·3 per cent. of British troops, and 16 per cent. of the Italians. *J. F. Corson.*

SPÜHLER, O. & KARTAGENER, M. Endemisches Auftreten eosinophiler Lungeninfiltrate in einer militärischen Einheit nebst Bemerkungen über das " miliare " eosinophile Infiltrat. [**Pulmonary Infiltration with Eosinophilia in a Military Unit.**] *Schweiz. med. Woch.* 1944, Nov. 4, v. 74, No. 44, 1145–9, 2 figs. [13 refs.]

The authors review papers which have been published on the presence of Loeffler's syndrome in association with *Ascaris* infestation, and give notes in tabular form of 28 patients, stating the X-ray appearances of the lungs, the percentage differential leucocyte counts, the presence or absence of *Ascaris* ova in the stools, and the *Ascaris* skin test.

All showed the pulmonary infiltration, but the eosinophilia was not very high, only one reached 35 per cent. *Ascaris* ova were found in the stools of 15, not found in 9 (but one of these had had treatment for ascariasis), in four cases no statement is made [perhaps the stools were not examined in these]. As regards the cutaneous test, in six it was positive and *Ascaris* ova were present ; in four it was positive but ova were not seen, in another four the test was negative and no ova were seen, in four the result of the test was negative, but ova were present ; in the others the test does not seem to have been carried out, at all events there is no record of it. In spite of these results being so much at variance the authors regard the syndrome as an allergy due to *Ascaris* infestation. [On their own showing in the table, there is as much evidence from these cases *contra* as *pro*]. *H. Harold Scott.*

CARDIS, F. A propos de l'infiltrat pulmonaire fugace avec éosinophilie sanguine et de son étiologie le plus souvent vermineuse. [**Transient Infiltration of the Lungs with Eosinophilia, often of Helminthic Origin.**] *Schweiz. med. Woch.* 1945, Feb. 24, v. 75, No. 8, 165–70. [34 refs.]

[This is a very intriguing article (using the adjective in its journalistic sense) for it is interesting, plausible, and may contain much truth. The author states the problem fairly and marshals his facts and arguments in an orderly fashion.] He starts by giving the characteristic clinical manifestations of Loeffler's syndrome : its benign character and its transitoriness, the slight systemic disturbance, little if any rise of temperature, slight cough, almost total absence of expectoration, and lack of physical signs. He next mentions the X-ray manifestations and the blood-changes, the chief part is devoted to a discussion of the diagnosis and possible aetiology, particularly a broncho-pneumonia, primary or post-influenzal, epituberculosis (but this is not transient, leaves a permanent scar and is not associated with eosinophilia) and atelectasis. The author then discusses the *ascaridosis theory* and *allergy*. Loeffler, when bringing his cases before the Swiss Tuberculosis Association in 1936, suggested that the condition might be a " pulmonary tuberculide ", a benign tuberculosis, but children and adults presenting the syndrome reacted negatively to the von Pirquet test and the infiltration did not proceed to true tuberculosis. Allergy was postulated to the sun's rays (WIELAND), to cold (LEITNER, and others), to various pollens, privet, lilies, cherry-laurel. MÜLLER, who himself suffered from the syndrome, attributed it to eating raw salads and brought on a recurrence by ingesting soil from his garden which contained *Ascaris* ova, and produced

the same effects in two of his fellow-workers. Thus *Ascaris* could experimentally set up the symptoms, but was it the only cause, and if so, how did it act? *Echinococcus* and *Fasciola hepatica* can produce some of the symptoms, but they cause pain in the liver, rise of temperature, dyspnoea.

Then came the work of VOGEL and MINNING who conducted systematic experiments with *Ascaris*. To six persons they gave *Ascaris* eggs, 6 to 45 in number. In five of these Loeffler's syndrome was produced and in four the worm was found later in the intestines. They made the following observations: The clinical and radiological signs of the syndrome appeared between the 6th and 12th days after infestation and lasted for three to eight days; the radiological opacities "appeared, developed and disappeared" between the 6th and 20th days after infestation; there was sometimes headache, with a sense of fatigue and heaviness of the limbs for 3–4 days; there was cough but no oppression in the chest, fever to 38·4°C. in one patient only; expectoration showed 80 per cent. of the cells to be eosinophiles; no *Ascaris* larvae were seen; blood eosinophilia ranged between 8 and 60 per cent. at its maximum from the 19th to the 21st days after infestation. There was observed also, at the time when the pulmonary signs disappeared, urticaria, discrete erythema, prurigo and dyshydrosis. Another fact noticed was that ova began to appear in the stools on the 68th day. This means that if we are to attribute the syndrome to *Ascaris* (eggs or larvae) we should look for ova in the stools between nine and ten weeks later. In other words, it is not the intestinal parasitism *at the time* which is the cause of the syndrome, and to prescribe an anthelmintic at the time of the syndrome is to deprive ourselves of an important source of confirmation later (*i.e.* by finding ova 9–10 weeks afterwards).

It has been thought that the pulmonary manifestations were due to the presence and passage of *Ascaris* larvae in the course of their development and life-history, but larvae are not seen in the sputum nor found in the lungs at autopsy. This is not to be wondered at, for the larvae do not come there to be eliminated in the sputum, but are "birds of passage" passing through the lungs as a stage in their normal development. The interstitial infiltration, the giant cells (foreign body type), the minute haemorrhages are more readily understood as mechanical rather than anaphylactic results of the presence of the larvae; moreover, the syndrome is not produced by intradermal injection of *Ascaris* antigen. In short, the syndrome, it is concluded, is pulmonary ascaridosis and this should be regarded as a "presumptive diagnosis" which can be confirmed by finding ova in the stools 9–10 weeks later, and inquiry should be made as to the patient's having eaten raw salads within the previous ten days [but few could deny this whether they presented the signs of Loeffler's syndrome or not]. *H. Harold Scott.*

LYON, E. & KLEINHAUS, E. M. **An Eosinophilic Disease with Cutaneous Manifestations associated with Transitory Pulmonary Infiltrations (Loeffler's Syndrome).** *Acta Med. Orientalia* (*Palestine & Near East Med. J.*) 1945, May, v. 4, No. 5, 144–9, 4 figs.

Twenty cases are mentioned as instances of Loeffler's syndrome and two are detailed, one a man of 43 years, the other a woman of 39. There was transitory pulmonary infiltration with eosinophilia, but the symptoms in other respects differed considerably from those regarded as characterizing Loeffler's syndrome. Thus, there was cutaneous oedema with redness of the skin, "resembling urticaria or erysipelas" and a swelling of the roof of the mouth lasting for three days, in one patient. In the other there was localized swelling on the right thigh as large as the palm; a return of the same a fortnight afterwards, and a swelling of the back with pain which persisted for over a month;

also a rise of temperature on the first and third occasions ; no mention is made of any rise on the second, nor subsequently when a patch again appeared on the thigh. In the first patient there was also a maculo-papular rash followed four days later by a diffuse tender swelling on the neck. Examination of the stools revealed no helminths or their ova. No statement is made in this respect in any of the other cases. [Obviously, these cases differed in many important respects from what has been reported elsewhere as Loeffler's syndrome.]

H. Harold Scott.

WRIGHT, D. O. & GOLD, E. M. **Loeffler's Syndrome associated with Creeping Eruption (Cutaneous Helminthiasis).** *J. Amer. Med. Ass.* 1945, Aug. 11, v. 128, No. 15, 1082–3. [Refs. in footnotes.]

During a period of twelve months the authors saw 12 cases of creeping eruption and observed fifteen of them continuously for a fortnight or more, taking X-ray photographs of the chest every three days. Nine of them presented the typical signs of Loeffler's syndrome—patchy migratory infiltration of the lungs, very little, if anything, in the way of physical signs, cyanosis or dyspnoea, and no fever, but with eosinophilia of the blood and sputum. These indicate, as others have also maintained, that the infiltration affects the interstitial tissue of the lungs, predominantly at least.

Total blood counts are not given, but the eosinophil percentage was up to 38 in the blood and to 90 in the sputum. Larvae of *Ancylostoma braziliense* are thought to be the cause in these cases, but no ova or worms were seen in 81 stools examined from the 26 patients. Infestation of the intestine by this nematode is known to be rare.

The Loeffler syndrome has now been reported in association with asthma, pollinosis, undulant fever, tuberculosis, amoebiasis and various helminthic infestations, *Ascaris*, *Trichinella* and others, and with acariasis. "Such a diversified list of etiologic factors lends credence to the view that Loeffler's syndrome is an allergic phenomenon," say the authors. *H. Harold Scott.*

GOTSHALK, H. C. **Transient Pulmonary Infiltrations (Loeffler's Syndrome). Report of a Case.** *Hawaii Med. J.* 1945, July–Aug., v. 4, No. 6, 302–4, 8 figs.

EARLE, K. V. **Vincent's Infection in Northern Peru.** *Trans. Roy. Soc. Trop. Med. & Hyg.* 1945, Sept., v. 39, No. 1, 77–82. [18 refs.]

Ulcerative stomatitis associated with Vincent's organisms is widespread in the desert region of northern Peru ; it is commonest in the *peon* [labouring] class but occurs also in the *empleado* [better paid employee ?] class. Among 403 patients admitted to hospital for various complaints, 241 had dental caries, 207 pyorrhoea, and 142 gingivitis. Swabs were taken from the mouths of 63 patients and Vincent's organisms were found in 54 ; these 54 patients were studied with regard to the presence of avitaminosis and debilitating diseases.

Avitaminosis A was indicated by Bitot's spots (2 cases), nyctalopia (7), phrynodermia (7), fine scaling (17), respiratory disorders (12), and enlarged and septic tonsils (24).

Thiamin deficiency was shown by various signs of neuritis (20), anorexia (10) and constipation (10). Evidence of ariboflavinosis was presented by cheilosis (5), scrotal dermatitis (1) and seborrhoeic disorders. Other probable signs of lack of vitamin B were coarse scaling of the skin (in 1·5 per cent.) hyperpigmented skin areas (34·47 per cent.) and hypopigmented skin areas (20·59 per cent.). Tongue changes included dryness and atrophy (3 cases), magenta colour (2), rawness, soreness, fissures and redness (7).

There were also dental changes which suggested avitaminosis. Debilitating conditions were caused by a variety of diseases.

The author remarks on the absence of tropical ulcer although many conditions thought to be aetiologically related to it were present, and suggests that the temperate and dry climate may be the reason ; a table of meteorological readings is given to show the difference of this climate from that of Trinidad, where tropical ulcer is common. *J. F. Corson.*

SHARP, H. S. & BIGGS, R. **A Case of Rhinosporidiosis.** *J. Laryngol. & Otol.* 1944, Dec., v. 59, No. 12, 457–8, 1 pl.

An Indian man, a native of Orissa, India, began to get nasal obstruction on the right side in 1933 ; he came to England in 1937 and, as the obstruction got worse, he attended a hospital in 1938 and had " polypi " removed. Since then he had had nine intranasal operations for recurrent polypi, the longest period of relief being 6 months. He was seen by the authors at Oxford in November 1944.

His right nostril showed several bleeding polypoid growths like pale granulation tissue, on which could be seen several small white spots ; there was a thin muco-purulent discharge. The growths were sessile on the septum just in front of the middle and inferior turbinates. They were removed by snare under local anaesthesia with galvanocautery to the base.

The tissue removed had the typical appearance and structure of the polypi caused by *Rhinosporidium seeberi*, large numbers of the parasite, in different stages of development, being present in the granulation tissue [see ASHWORTH, this *Bulletin*, 1923, v. 20, 451 ; KARUNARATNE, *ibid.*, 1936, v. 33, 720].

J. F. Corson.

GENERAL ENTOMOLOGY.

LEWIS, D. J. **Observations on the Distribution and Taxonomy of Culicidae (Diptera) in the Sudan.** *Trans. Roy. Entom. Soc. London.* 1945, June 30, v. 95, Pt. 1, 1–24, 2 maps. [22 refs.]

The paper is a summary of our knowledge of the mosquitoes of the Sudan. The total number of species now known to occur is 127, including 26 anophelines. The map shows that specimens have been collected from almost all parts of the territory, including isolated mountains and oases in the west ; the more fertile south-west has apparently not been very fully studied.

The author calls attention to several interesting points in zoogeography. It has been known for some years that the range of certain Mediterranean mosquitoes extends down the Red Sea into the arid Somali coast, but it now appears that some of these northerners also extend their range much farther west, even to Darfur. Many of the geographical distributions are remarkably discontinuous, and mosquitoes are recorded from certain remote areas surrounded by desert : for instance *Anopheles cinereus* is found in Jebel Marra, and *Aëdes caspius* in that very isolated spot the Merga oasis. Isolated and discontinuous range such as this (and other examples which might be quoted from the Sahara) are probably due to a much more continuous distribution in a former more rainy epoch.

Among insects of medical interest one observes that *Anopheles gambiae* is found all along the Nile throughout its course through the Sudan, and more widely in the southern and central Sudan. *Anopheles funestus* occurs along

the larger rivers, northwards to about 13°N. also in the Nuba mountains ; the distribution of this species and the closely similar *A. rivulorum* and *macmahoni* is shown on a map. *Aëdes aegypti* has a very wide distribution, and on the coast is generally represented by a variety in which the normally black areas of the thorax are brown or creamy, though the sharply contrasted black and silver legs remain as in the type. It is a matter of interest that *Culex andersoni*, not otherwise known to occur in the Sudan, has been collected in Khartoum in aircraft coming from Asmara and from Addis Ababa. *A. gambiae* has been found in a train arriving at Port Sudan. *P. A. Buxton.*

CAMBOURNAC, F. J. C. Culicidae (Diptera, Nematocera) da região de Aguas de Moura. [**The Culicidae of the Region of Aguas de Moura.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 247–68, 2 pls. [18 refs.]

THEODOR, O. H. & PARSONS, B. T. **Insectary Rearing of** *Anopheles pharoensis*. *Bull. Entom. Res.* 1945, July, v. 36, Pt. 1, 79–83.

A colony of *Anopheles pharoensis* was maintained for three generations in the neighbourhood of Cairo between September 1943 and January 1944. This paper describes the methods employed and the results achieved.

The breeding cage was adapted from that described by HACKETT & BATES at the Amsterdam malaria congress in 1938 [see *Rev. Applied Entom.*, Ser. B, 1940, v. 28, 84]. The mosquitoes were fed on sugar and water solution in a small sponge suspended from the cage ceiling ; blood meals were obtained from a rabbit at first, subsequently human blood was provided by putting the fore-arm into the cage once a day. A constant temperature could not be maintained ; humidity was provided by a damp blanket ; light entered through the glass top and each evening a blue " swarming light " was switched on. Eggs laid in Nile water in Petri dishes were removed daily to white enamel bowls. After some experimentation, first-stage larvae were fed on a suspension of bakers' yeast ; from second stage onwards they were fed on finely ground army biscuit. Larvae were exposed to the sun daily for about eight hours. Mean monthly temperatures in the insectary gradually fell from 28·5°C. in September to 13·7°C. in January. On the approach of colder nights attempts to raise the temperature by means of an oil stove failed, mainly because the stove could not be left burning all night. Larvae were therefore kept in an incubator overnight, first at 23°C. and later at 27°C. The duration of the larval period at these temperatures (nightly) was twenty-three and eighteen days respectively. The pupal period was 3–4 days at 20°C. The resulting larvae, pupae and adults were as big and vigorous as in natural habitats.

The colony was started with mosquitoes bred from locally collected larvae and 260 adults were introduced on 31st October to stimulate swarming. By 22nd November 1,038 males and 1,106 females were produced. From this time till 31st January 1,126 males and 1,081 females were entirely laboratory bred. The last batch of eggs was laid on 19th January. The authors believe that with better facilities for maintaining a suitable temperature in the insectary during the cold weather, these figures could be much improved and it was only the lack of these facilities that ended the experiment.

H. S. Leeson.

DE MEILLON, B., GOLBERG, L. & LAVOIPIERRE, M. **The Nutrition of the Larva of** *Aëdes aegypti* **L. I.** *J. Exper. Biol.* 1945, Aug., v. 21, Nos. 3 & 4, 84–9.

GOLBERG, L., DE MEILLON, B. & LAVOIPIERRE, M. **The Nutrition of the Larva of** *Aëdes aegypti* **L. II. Essential Water-Soluble Factors from Yeast.** *J. Exper. Biol.* 1945, Aug., v. 21, Nos. 3 & 4, 90–96. [20 refs.]

DE MEIRA, M. T. V. & FERREIRA, T. G. Espèces de phlébotomes de Lisbonne et de ses environs. [**Species of *Phlebotomus* in and around Lisbon.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 269–88, 5 pls. (1 map) & 2 figs. English summary. [31 refs.]

FERREIRA, F. da C. & FERREIRA, T. Sôbre a biologia das espécies de *Phlebotomus* de Lisboa e Arredores. [**The Biology of the *Phlebotomus* Species of Lisbon.**] *An. Inst. Med. Trop.* Lisbon. 1944, Dec., v. 1, No. 2, 289–313, 5 figs. & 1 pl. [41 refs.] English summary.

BROWN, E. H. **Screwworm Infestation in the Nasal Passages and Paranasal Sinuses.** *Laryngoscope.* 1945, July, v. 55, No. 7, 371–4. [10 refs.]

LINDQUIST, A. W., SCHROEDER, H. O. & KNIPLING, E. F. **Concentrated Insecticides: Preliminary Studies of the Use of Concentrated Sprays against Houseflies and Mosquitoes.** *Soap.* New York. 1945, July, v. 21, No. 7, 109, 111, 113 & 119.

This paper sets out primarily to answer the question " would 1 ml. of a fly spray containing 10 per cent. of toxic ingredients be as effective as 10 ml. of spray containing 1 per cent. of toxic ingredients, provided that the concentrated sprays were efficiently atomized ? " The authors conclude that this would in fact be the case.

Two insects, the house fly *Musca domestica* and the mosquito *Anopheles quadrimaculatus*, were used in the tests. The insects were exposed in cages which were carried through the treated rooms. The mosquitoes were exposed for one minute and the house flies for five minutes. Pyrethrins and 2,2 bis (parachlor phenyl) 1,1,1 trichlorethane (DDT) were employed as the toxic agents, while the solvent was kerosene supplemented when necessary by some more effective ingredient (unnamed). The concentrated sprays were also compared with dichlorodifluoromethane (Freon-12) aerosols, using equivalent dosages of toxic ingredients in the air space.

From the point of view of economy of solvent and transport, concentrated sprays have obvious advantages. When finely atomized as with a De Vilbiss paint spray gun operated at 25 lb. per sq. in., equal quantities of DDT, or pyrethrins, or mixtures of the two, gave similar knockdowns and kills, whether dispersed from concentrated (*e.g.* 16 per cent. DDT) or dilute (*e.g.* 1 per cent. DDT) solutions. These concentrated, finely dispersed sprays also compared favourably with Freon aerosols when tested biologically. When houseflies were exposed in rooms treated with 4 mgm. pyrethrins and 40 mgm. DDT per 1,000 cu. ft. for five minutes, and the exposure was commenced even thirty minutes after spraying, kills in the region of 85–95 per cent. resulted from both the aerosol and concentrated spray applications. In the latter case the spray was applied by a pocket-size capillary-tube sprayer specially designed to distribute these concentrated sprays. In practical terms such a sprayer, if it contained 75 ml. of concentrated spray, would be capable of treating 150,000 cu. ft. of space, which would require approximately 2¼ quarts of " Official Test Insecticide " (0·1 per cent. w/v pyrethrins) if finely atomized, or about one gallon if dispersed through an ordinary hand sprayer. *W. A. L. David.*

STARR, D. F. **Use of a Double-Nozzled Spray Apparatus for the Application of DDT or Oils.** *Science.* 1945, Aug. 10, 156–7.

By mixing two sprays, one of DDT solution and the other of water, a deposit on the sprayed surface was obtained which was superior in quantity and quality to that obtained by using a spray of DDT solution alone. The DDT

solution was sprayed from a small atomizer and the water spray was from a suction type paint sprayer. The two nozzles were set to converge at an angle of 35° and the two sprays met at 2 to 4 cm. from the nozzles.

When the DDT solution was made with water-miscible solvents such as acetone, dioxane or alcohol, the DDT was deposited as "nascent precipitates," some liquid and some solid; when solvents not miscible with water, such as oil, were used, the solvent (oil) remained on the sprayed surface while the water drained off and a heavy deposit of oil could be obtained without losing much in the water draining off.

The deposits of DDT on glass were extremely resistant to water dropped on them like rain; some nascent deposits showed no decrease in amount after 7 hours of such treatment.

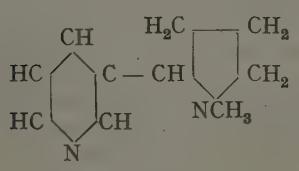
Oranges and apples were successfully sprayed by this method.

J. F. Corson.

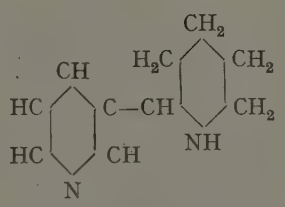
GEAR, H. S. **A Note on the Use of D.D.T. in Medical and Health Problems.** *South African Med. J.* 1945, Aug. 25, v. 19, No. 16, 290–92.

NABOKOV, V. A. **Anabasine Sulfate: a Protective Agent against Bites of Malarial Mosquitoes.** *Amer. Rev. Soviet Med.* 1945, June, v. 2, No. 5, 449–52.

A useful repellent for mosquitoes was found in the substance anabasine sulphate prepared from the plant *Anabasis aphylla* (Chenopodiaceae). Anabasine, which is the chief alkaloid present (55 per cent. of total alkaloids), has a formula somewhat similar to nicotine, thus :—



Nicotine



Anabasine

Anabasis is common in the U.S.S.R. and the alkaloids in the form of sulphates have been widely used as insecticides. Undiluted anabasine sulphate is a dark brown, oily liquid with a characteristic irritating odour. It is soluble in water and alcohol, giving an acid reaction. The 5 per cent. solution is almost odourless and can be wiped off the skin without difficulty.

Aqueous solutions of 1, 3, 5 and 10 per cent. were tested for mosquito repellent powers. It was found that none of them greatly diminished the numbers of mosquitoes alighting and that 1 and 3 per cent. had no effect in preventing bites. However, with 5 per cent. and still more with 10 per cent., there was a very high degree of protection from bites. This protection lasts for five to six hours and with two applications complete protection is ensured for ten to eleven hours.

The possibility of dermatitis was investigated by patch tests. A 50 per cent. alcoholic or aqueous solution applied for four days gave some reaction (erythema with minute follicular nodules) but the application of 5 per cent. solution twice daily for a month to face, neck, chest and arms did not produce any harmful effect on the author.

Anabasine, like nicotine, can be absorbed through intact skin and can produce systemic toxicity. It is, however, considerably less toxic than nicotine; 0·1 mgm. of nicotine being equal to 0·5 mgm. anabasine or 1 mgm. lobeline. Anabasine sulphate is much less toxic than the base and should not be dangerous when used diluted. *J. R. Busvine.*

LABORATORY AND TECHNICAL PROCEDURES.

GILLMAN, T. & GILLMAN, J., with the technical assistance of J. G. BRYDEN. **A Modified Liver Aspiration Biopsy Apparatus and Technique, with special reference to its Clinical Applications as assessed by 500 Biopsies.** *South African J. Med. Sci.* 1945, June, v. 10, No. 2, 53–66, 1 text fig. & 5 figs. on 1 pl. [26 refs.]

The operation of liver puncture has not hitherto been free from danger to life, as from 1 to 2 per cent. of patients have been killed by it, usually from haemorrhage due to tearing of the liver tissue by the needle. In a proportion (22·5 and 50 per cent.) of cases, also, the operation has failed, liver tissue not having been obtained.

The authors have modified the syringe and needle of IVERSEN and ROHOLM (*Acta Med. Scandinavica*, 1939, v. 102, 1) in several particulars: the stylet remains in the cannula, so preventing the liver tissue from being drawn into the syringe; the back of the plunger has a rubber buffer; the end of the cannula is bevelled and makes a slit wound instead of a gaping round hole in the liver; the shaft of the plunger is locked in position when the plunger is pulled back, so preventing the tissue in the cannula from being accidentally expelled by recoil of the plunger.

Patients fast for 12 hours before the operation. If the liver is enlarged or if there is jaundice the prothrombin level is determined and vitamin K given if required. Morphine is only necessary in very nervous patients or if the liver is very tender.

If the liver is much enlarged the patient lies flat in bed with arms extended above his head; in other cases he is propped up in a semi-sitting position. Babies are dealt with in the semi-sitting position.

The skin and underlying tissues (including the peritoneum) in the apex of the angle between the xiphisternum and the right costal margin, are successively anaesthetized with 2 per cent. novocain, in a direction slightly upward and to the left. Anaesthesia is established in 2–3 minutes, then the skin is slightly incised and the needle, attached to the syringe, is pushed upwards, backwards, and slightly to the left until the surface of the liver is felt, when the "depthguard" is quickly adjusted to permit the needle to go only 2–3 cm. further. The needle is pushed into the liver and the plunger quickly withdrawn and the barrel quickly twisted through 360 degrees on the plunger and the instrument withdrawn. The whole operation of puncture should take less than 15 seconds.

The method was tested first on baboons and human corpses and has since been used in more than 500 biopsies on 260 human subjects and in more than 150 on baboons and other monkeys, without shock or haemorrhage, and only one death; in that case haemorrhage was due to puncture of an unusual artery in the liver and not to tearing of liver tissue. The authors have used

it in normal subjects (20), cases of pellagra (92 infants and 349 adults), carcinoma of the liver (16), cirrhosis of the liver (10), hepatitis, tuberculosis, schistosomiasis, malaria and diabetes. In diffuse lesions it is reliable, but in localized lesions positive results only are significant.

The authors refer to their results (some still in the press) and to the work of ROHOLM and IVERSEN (*Acta Path. Microbiol. Scandinavica*, 1939, v. 16, 427) and of DIBLE, MCMICHAEL and SHERLOCK (*Bulletin of Hygiene*, 1944, v. 19, 14) and mention other applications of this method of liver puncture. [See also this *Bulletin*, 1944, v. 41, 1057, 1058.] *J. F. Corson.*

SHERLOCK, Sheila. **Aspiration Liver Biopsy. Technique and Diagnostic Application.** *Lancet.* 1945, Sept. 29, 397–401, 6 figs. [22 refs.]

The technique of aspiration liver biopsy is described and the risks involved and its use in diagnosis are discussed.

A jaundiced patient should be given vitamin K for 3 days before the puncture is made; in non-obstructive jaundice (urobilin in the urine) two 5 mgm. tablets of "Kapilon" should be given by mouth three times a day, while in obstructive jaundice a daily dose of 5 mgm. should be injected intramuscularly. The patient's blood group must be known and two pints of compatible blood must be ready for transfusion if required. A sedative before puncture is not necessary.

The patient lies supine in bed with his right side near the edge and his right arm placed behind his head; a firm pillow may be placed under his left side to tilt his body slightly to the right.

The cannula is 15 cm. long and 1 mm. in bore and is fitted with a handled trocar; a 20 cc. "record" syringe is used.

The puncture is made in the mid-axillary or anterior axillary line in the 9th or 10th intercostal space [*cf.* GILLMAN and GILLMAN, above]. The skin is anaesthetized with 2 per cent. procaine [novocain] solution and by means of a long fine-bore needle the pleura and peritoneum and liver capsule are also anaesthetized, at least 10 cc. of solution being required.

The trocar and cannula are passed through the skin (which may be previously incised) and the patient is told "take a deep breath in, let it out, and then hold your breath." This displaces the lung upwards and ensures apposition of the diaphragmatic and costal pleurae. The trocar and cannula are then pushed into the liver, and when it is fully half an inch within the liver, the trocar is withdrawn and the cannula pushed in for a further 4–5 cm. to punch out a cylinder of liver tissue; the syringe is then attached and suction maintained while the cannula is withdrawn. The puncture wound is sealed with collodion. The liver fragment is usually found in the barrel of the syringe [see GILLMAN & GILLMAN above].

Afterwards a small dose of morphine may be given to relieve pain and in the evening a sedative such as barbitone soluble, 10 grains. The pulse rate should be charted hourly for the first 24 hours and if it rises, the physician should be called. Routine visits should be made 4 and 8 hours after the biopsy. If there is any sign of haemorrhage blood transfusion should be done. Absolute rest in bed for the first 24 hours is essential, but the patient may then get up and leave the hospital 48 hours after the puncture.

Difficulties.—The author and her colleagues [see DIBLE, MCMICHAEL and SHERLOCK, *Bulletin of Hygiene*, 1944, v. 19, 14] failed to obtain a suitable specimen of liver tissue in 10 per cent. of their first 126 biopsies but in only 2 per cent. of the next 138. Difficulties arise chiefly in hepatic cirrhosis, especially if ascites is also present; also, in pulmonary emphysema the liver is pushed downwards and the cannula may easily pass above it.

Risks.—Fatality rates in the past varied from 0 to 3 per cent. (a table is given); in the present series of 264 punctures there were two deaths (one patient was already moribund) which occurred in the first 126 cases [see DIBLE *et al. loc. cit.*]. This decrease in risk is due to changes in technique and a more careful selection of cases. The bore of the cannula was reduced from 2 mm. to 1 mm. and the length increased from 10 cm. to 15 cm. which enables the capsule of the liver to be cut cleanly by the trocar. The risk of haemorrhage is greatest in severely jaundiced patients especially in acute parenchymatous liver disease; in the former series (126 cases) haemorrhage occurred in 5 out of 83 cases with jaundice, in the latter (138 cases) in none out of 74.

Except in localized lesions the information obtained by this method is reliable; sections of 10–20 liver lobules can be cut from the piece removed. Diseases in which it is useful for diagnosis are: acute hepatitis, cirrhosis of the liver, obstructive jaundice, malignant disease of the liver, blood diseases, amyloid disease, kala azar, and various other diseases; notes on some cases are given. The method may also be used to test the action of drugs on the liver. *J. F. Corson.*

FULLER, A. T. **The Estimation of Aromatic Amidines.** *Biochem. J.* 1945, v. 39, No. 1, 99–102, 2 figs.

The principle of the estimation is that when solutions of aromatic amidines are heated with glyoxal sodium bisulphite and boric acid at *p*H 9 a colour (usually magenta) is produced which can be measured in the usual ways. The reaction is specific for an unsubstituted amidine directly joined to an aromatic nucleus; other amidines cannot be measured in this way. Thus the method can be used for stilbamidine, propamidine, and V.187 (*p*-methyl sulphonyl benzamidine), although with the first two of these the method is not sensitive enough to measure therapeutic blood concentrations. The compounds can be estimated in blood, urine, and faeces. The exact instructions for carrying out the estimation vary somewhat with the different compounds, their concentrations and the biological fluid in which they are present; for these technical details, the original paper should be consulted. *F. Hawking.*

REPORTS, SURVEYS AND MISCELLANEOUS PAPERS.

FAN, J. H. **Communicable Diseases in China during Recent Years.** *Epidemiological Information Bull.* (*UNRRA Health Division*). Washington, D.C. 1945, July 31, v. 1, No. 12, 495–536, 1 map. [38 refs.]

This is a detailed summary of reports on the occurrence, during the years 1939–44, of seven of the ten notifiable diseases in China; they are: plague, smallpox, typhus, relapsing fever, cholera, dysentery and typhoid fever (including paratyphoid fevers).

Plague.—During recent years plague has become more prevalent and has spread inland from the south-eastern coastal regions. In 1940 the Japanese dropped rice and wheat mixed with rat-fleas over the city of Chu-hsien and probably over Ning-po, and plague appeared for the first time in these cities soon afterwards and is thought to have been caused in this way. There were

11,729 cases of plague in ten provinces during the period, those chiefly affected being Fukien and Chekiang ; in the former the incidence rate averaged 16 per 100,000 of the population, and in the latter 23·4 per 100,000.

Smallpox.—Localized outbreaks still occur, especially in connexion with migrations of population, and in 1941 and 1942 there was a widespread epidemic. In 19 provinces 38,836 cases were reported, the average rate of incidence being 2·4 per 100,000 of the population.

Typhus.—In North China, where it is the most prevalent, it corresponds clinically and epidemiologically, according to many authors, to the epidemic type, but murine flea-borne or endemic typhus has been proved to exist in Peiping and was reported from Shanghai. In 19 provinces 23,130 cases were reported, an average incidence of 1·8 per 100,000.

Relapsing fever.—This is louse-borne in China ; 72,845 cases were reported in 19 provinces, the average incidence being 4·7 per 100,000. In certain Chinese armies there were 12·3 cases per 1,000 in 1939 and 7·4 in 1940, among about half a million men in each year.

Cholera.—During the period, 92,216 cases (5·5 per 100,000) were reported in 19 provinces.

Dysentery.—It is estimated that bacillary dysentery formed about 90 per cent. of the total cases reported ; there were 361,361 cases (25·9 per 100,000) in 19 provinces. In certain Chinese armies rates of incidence of 12·8 and 8·7 per 1,000 occurred in 1939 and 1940 respectively.

Typhoid fever.—In North China paratyphoid A, B and C fevers are said to be prevalent as well as typhoid fever ; 64,097 cases (4·6 per 100,000), including paratyphoid, were reported during the period.

The spread of plague and of some of the other diseases is discussed in some detail and figures of monthly incidence in the counties of the provinces are shown in tables ; a map is given and there are 38 references.

J. F. Corson.

WICKRAMASINGHE, W. G. **A National Health Service for Ceylon.** *J. Ceylon Branch Brit. Med. Ass.* 1945, Apr., v. 41, No. 1, 1–12.

BOOK REVIEWS.

LEE, David J. [B.Sc.] & WOODHILL, A. R. [B.Sc. Agr. (A.A.M.C., Australia)]. **The Anopheline Mosquitoes of the Australasian Region.** Publications of the University of Sydney, Dept. of Zoology. Monograph No. 2, pp. xii + 209, 34 pls., 8 maps & 13 figs. 1944, Glebe, N.S.W. : Australasian Medical Publishing Company Ltd., Seamer Street.

As a result of some two years' collaboration, and with the help of the Mobile Entomological Units, the Malaria Control Units and other members of the Australian Army Medical Corps, the authors have produced this concise review of existing knowledge on the Australasian anophelines. The region covered includes Australia, Tasmania, and islands eastward to 180° longitude ; New Guinea and islands northward to the equator, and the Moluccas, Ceram and Timor.

In the introduction, general matters concerning morphology, biology and control are briefly discussed. EDWARDS's classification is followed in all but minor respects; one departure is the adoption of the recent American custom of trinomial nomenclature for subspecies, a practice which the authors discuss in the section on "Systematics".

Special attention is paid to the characters used for the identification of adults and larvae in the account of the external morphology of the tribe *Anophelini*, taking *Anopheles annulipes* as the basis for comparison. The main body of the work, comprising some 130 pages, consists of detailed descriptions of the known males, females and larvae of about forty species and varieties; but in only six are eggs described, and pupae in none. There are keys to the species, notes on synonymy, type localities, biology and relation to disease. Closely related species and forms are compared, particularly the *A. punctulatus* complex, which comprises at least two, probably three and possibly more subspecies. The subspecies *punctulatus* and *moluccensis* are the principal dangerous vectors of malaria in the Australasian region; both occur in the Moluccas, New Guinea, New Ireland, the Solomons and other islands, where they form the dominant element of the anopheline population, but *moluccensis* occurs also on the mainland in Northern Australia and Queensland.

In addition, in less detail, short accounts of twelve other anophelines which occur on the western fringe of the Australasian region are appended. Three of them, *A. minimus*, *A. maculatus* and *A. sundaicus*, important vectors of malaria elsewhere, are recorded from Timor.

Recognition of the main distinguishing characters of the Australasian species and their distribution is greatly assisted by the clear line drawings in plates, text figures and maps. References are given to 120 papers and journals (some of them in the Dutch language) and an index is provided.

This much-needed, up-to-date monograph fills another gap on the library shelves of the medical entomologist, though revision will be required as knowledge increases. The control of mosquitoes does not come within the scope of this publication, but attention is drawn to some of its entomological aspects. Medical officers, malariologists and others will certainly gain much information regarding variations in morphology, biology and habits of these species of *Anopheles*, which will enable them to adapt their control measures to local circumstances. *H. S. Leeson.*

TROWELL, H. C. [M.D., F.R.C.P. (Lond.), etc.]. **Diagnosis and Treatment of Diseases in the Tropics.** Baillière's Medical Manuals for Africans. 2nd Edition. pp. xiv + 219, 35 figs. 1945. London: Baillière, Tindall & Cox, 7 & 8 Henrietta Street, Covent Garden, W.C.2. [4s.]

Writing this book must have been an undertaking far from easy. It necessitated a thorough knowledge of the mind of the better educated native African, of his capabilities of absorbing instruction and of applying the knowledge imparted; there was the additional difficulty of deciding what to teach and what to leave out in order to produce a competent dresser and nurse, one able to undertake on emergency even greater responsibilities, but not to overpass this boundary so that the student would come to think that he was able to act as a qualified doctor and assume greater responsibility than his instruction warranted.

This task could not have been entrusted to better hands. The author possesses the highest medical qualifications obtainable; he has been for some years in close touch with East Africans and engaged in instilling into them the elements of medicine and surgery.

The book is clearly intended to supplement lectures and notes and not to replace them. Thus, to give a single example, the instruction to "relieve pain by opium and morphia," or in the case of smallpox to "treat the case by nursing, fluid diet, opium and morphia" is too vague, if no dose or method of administration is mentioned.

There is little to add to what was written by the reviewer of this book when it was first published in 1939 [see this *Bulletin*, 1939, v. 36, 876], for the differences between that and the present issue are small. A few words here and there have been added on the use of one or other of the sulphonamide group of drugs, and a chapter on Diets and Malnutrition. This last was an important omission from the first edition and has now been remedied and the work has been done well. The fact, often forgotten or neglected, that "in disease the body requires just the same amounts of food as in health" recalls the dictum of a bygone physician (was it not Graves ?) who wished to have as his epitaph the words "He fed fevers."

Apart from the changes referred to, the new edition seems to be merely a reprint of the old, even the same misprints (very few, it is true) remain and the Index is the same except for the single addition of M. & B. The print is not so clear as in the first issue and the blocks show signs of wear, but these are probably inevitable in a war-time production. There are two or three matters omitted which one would have expected to see, and here and there directions and statements might be more clearly expressed. Thus, under Pneumonia, the danger of heart failure and sudden syncope is not, in the reviewer's opinion, sufficiently indicated. Death in pneumonia is due, in a large majority of cases, to the stress on the heart. To say that "Heart failure . . . should be prevented by resting the patient" is not enough ; mere raising in bed or turning unassisted may result in stoppage of a heart whose burden is already almost more than it can bear. On the same subject a question is asked : "What is the usual treatment of pneumonia as regards . . . diet ?" Looking up the text for answer all we find is : "The diet is according to the patient's appetite," unless the harassed native student applies the instruction given a few lines above ; "The patient must be fed and helped with the bedpan." A similar instance of Horace's *Brevis esse laboro, obscurus fio* occurs on p. 120, where the treatment of intestinal haemorrhage in typhoid fever is given as "stopping all feeding and morphia injection." On p. 61, on the treatment of Sprains, we find : "The sprained joint should not be used much for at least a week or two, possibly for longer," followed immediately by the words "an injection of procaine into the strained ligaments . . . allows the patient to use the affected joint immediately." The administration of sulphaguanidine in dysentery, of gentian violet for threadworm infestations and of mepacrine in malaria shows that the information has been generally brought up to date. On the other hand the use of serum in cerebrospinal fever is not even referred to [has it been entirely superseded ?] and though syphilis and syphilitic aortitis are spoken of, aneurysm is omitted from the causes of cough, in spite of its being troublesome and of characteristic type. Of the Cestodes *Taenia saginata* is considered, but nothing is said of *T. solium* or of hydatid. Yaws and gangosa are spoken of, but we find nothing about goundou or juxta-articular nodules, except in the case of the former, the remark that "yaws or syphilis may attack the bones of the nose and the roof of the mouth" ; myiasis receives no mention. Smallpox is spoken of, and rightly, as a very serious disease, but there is no reference to alastrim. Droplet infection is explained on p. 134, but elsewhere infection so conveyed is designated "inhalation of sputum." The frequent advice to apply hot fomentations would lead the student to infer that some fomentations are applied cold.

Most of the above remarks deal, it is true, with minor points, but it is these so-called minor points which might have been corrected when the new edition was called for. At the same time they cannot be said to detract from the intrinsic value of a good piece of work which should prove most useful and instructive to those for whom it has been specially written. The East African student is indeed fortunate in having so sound a teacher as Dr. Trowell.

H. Harold Scott.

MEDICAL RESEARCH COUNCIL. **The Medical Use of Sulphonamides.** By various authors. (Edited by F. HAWKING & F. H. K. GREEN). 2nd Edition. *M.R.C. War Memorandum No. 10.* 71 pp. 1945. London: H.M. Stationery Office. [1*s.* 3*d.*]

Over 50,000 copies of the first edition of this Memorandum [this *Bulletin*, 1943, v. 40, 867] have been sold in two years. The new edition, which has increased in length by some 30 pages, retains all the valuable features of the original, but has been considerably amplified in the light of subsequent experience. There are numerous minor additions, but the authors' main task has been to give accounts of new sulphonamide compounds, and to adjust the therapeutic methods recommended to the new situation created by the advent of penicillin. Among the former only "Sulphamerazine," phthalylsulphathiazole and "Marfanil" are described fully, although other new drugs are more briefly mentioned in connexion with the treatment of particular infections. Penicillin is dealt with by a new section on the relation of sulphonamide to penicillin therapy, which outlines their separate spheres and discusses their respective merits for the treatment of many conditions in which these spheres overlap. The stronger indications for penicillin are also emphasized in subsequent sections treating of individual infections and diseases, but its dosage and the technique of administration are not discussed. The first edition was written as a guide to the use of sulphonamides "in the light of their present or potential availability in Great Britain." The supply position for most sulphonamides has greatly improved, but the "availability" of penicillin presents far more serious problems than that of, say, sulphathiazole two years ago, and the authors have therefore been commendably cautious, retaining full instructions for sulphonamide treatment even in conditions in which penicillin is the treatment of choice. There are extensive sections on the treatment of wounds and burns, and an interesting discussion of group prophylaxis as applied to cerebrospinal fever, dysentery, and streptococcal infections of the upper air passages. As before, numerous references, mainly to recent work, are given in the text. The influence of this publication in rationalizing sulphonamide treatment must already have been very great, and the present edition should maintain and extend it.

L. P. Garrod.

Printed under the authority of HIS MAJESTY'S STATIONERY OFFICE
by The South Essex Recorders, Ltd., Ilford.

(725) Wt. P11/296 1/46 S.E.R.,Ltd. Gp. 432

INDEX OF AUTHORS OR SOURCES.

(The bracketed abbreviations after the page numbers indicate the subjects. Page numbers within brackets indicate papers not summarized.)

Am.	signifies	Amoebiasis and Intestinal Protozoal Infections.
Bart.	,,	Bartonellosis.
Bl.	,,	Blackwater.
B.R.	,,	Book Review
Chl.	,,	Cholera.
Def. Dis.	,,	Deficiency Diseases including Epidemic Dropsy.
Den.	,,	Dengue and Sandfly Fever.
Der.	,,	Dermatology and Fungous Diseases.
Dys.	,,	Dysentery (Bacillary and Unclassed).
Ent.	,,	General Entomology.
Haem.	,,	Haematology.
Heat Str.	,,	Heat Stroke and Allied Conditions.
Hel.	,,	Helminthiasis.
Lab.	,,	Laboratory Procedures.
Leish.	,,	Leishmaniasis.
Lep.	signifies	Leprosy.
Mal.	,,	Malaria.
Misc.	,,	Miscellaneous.
Misc. Dis.	,,	Miscellaneous Diseases.
Oph.	,,	Tropical Ophthalmology.
Pl.	,,	Plague.
Prot.	,,	General Protozology.
Rab.	,,	Rabies.
R.F.	,,	Relapsing Fever and other Spirochaetoses.
Reports, etc.	,,	Reports, Surveys and Miscellaneous Papers,
Sp.	,,	Sprue.
Tryp.	,,	Trypanosomiasis.
Typh.	,,	Fevers of the Typhus Group
Ulc.	,,	Tropical Ulcer.
Vms.	,,	Venoms and Antivenenes.
Y.F.	,,	Yellow Fever.
Ys.	,,	Yaws.

A.

Abdel Azim, M., with Barlow, 1017 (Hel.)
Abraham, A. C. & Samuels, R. D., 619 (Mal.)
Abramova, S. G., 642 (Pl.)
d'Abrera, V. St. E., 337, 959 (Mal.)
——, with Carter & Wedd, 73 (Ent.)
Accart, R., with Gorsse, 395 (Hel.)
Adams, A. R. D., 559 (Am.)
Addis, C. J. & Chandler, A. C., 302 (Hel.)
Adhicary, B. M., 434 (Mal.)
Adler, S. & Pulvertaft, R. J. V., 412 (Prot.
Afonsky, N. P., 257 (Bl.)
Africa, Méd., 762 (Misc.)
Afrique Occidentale Française, 275, 723 (Y.F.), 289 (R.F.), (326) (Reports, etc.), 678 (Rab.)
Ahmad, N., 222 (Haem.)
Ahuja, H. L., with Bose, 121 (Chl.)
Ahuja, M. L. & Brooks, A. G., 830 (Vms.)
Aitken, G. J., with Paulley, 55 (Def. Dis.)
Alcantara, A. G., 988 (Typh.)
Alexander, J. S., Park-Ross, M. & Stein, M., 38 (Am.)
Alexander, R. S., with Warin, 380 (Dys.)
Alexander-Jackson, E., 899 (Lep.)
Alicata, J. E. & Breaks, V., 271 (Typh.)
Alisov, P. A. & Kossovaja, E. M., 989 (Typh.)
Allen, A. S., 69 (Misc. Dis.)
Allen, C., 253 (Mal.)
Allen, H. C., 49 (Hel.)
Allen, R. R., with Baer, 419 (Reports, etc.)
Allen, S. D. & O'Brien, J. P., 313 (Heat Str.)
Aller Atucha, J. F., 902 (Lep.)
Alli, J. H., 937 (Prot.)
Allison, D. K., with Kessel, Kaime, Quiros & Gloeckner, 40 (Am.)
Allison, J. R., 582 (Def. Dis.)
de Almeida, E., 876 (Leish.)
de Almeida Prado, A., 143 (Der.)
Almy, T. P. & Harper, J. G. M., 646, 815 (Hel.)
Alston, J. M. & Broom, J. C., 210 (R.F.)
Altmann, A., 652 (Haem.)
Aluimov, A. Y., with Pavlovskiĭ, 565 (R.F.)
Alvarado, C. A. & Peña, L. S., (448) (Mal.)
Alves, W., 815 (Hel.)
Amendola, F., 901 *bis* (Lep.)
Anderson, A. B., with Brown, 1023 (Haem.)
Anderson, C. R., 106, 192 (Typh.)
Anderson, G. A., von Deschwanden, J., Gray, W. A. & Menzies, T. H., 961 (Mal.)
Anderson, H. H., with Chen & Zia, 734 (R.F.)
—— & Chuan, T. T. K., 563 (Am.)
Anderson, M., with Grant & Thompson, 187 (Tryp.)
Anderson, T. F., Begg, F. H. H. & McNaughton, F., 497 (Misc. Dis.)
Andreasen, A. T. & Suri, H. L., 742 (Hel.)
Andreev, L. A., 898 (R.F.)
Andrewes, C. H., King, H., van den Ende, M. & Walker, J., 20 (Typh.)
Andrews, H. L., with Brady, Lawton, Cowie, Ness & Ogden, 747 (Hel.)
Andrews, J. C. & Cornatzer, W. E., 534 *bis*. (Mal.)
Anduze, P. J., 322 (Ent.)
——, with Hecht, 465, 640 (Y.F.)
Anfinsen, C. B., with Ball, Geiman, McKee & Ormsbee, 867 (Mal.)
Angel Abalo, M., with Leon y Blanco, 225 (Der.)
Angevine, D. M., Hamilton, T. R., Wallace, F. G. & Hazard, J. B., 976 (Leish.)
Anguiano, L. G., with Del Pozo & González, 653 (Vms.)
Anigstein, L., (229) (Ent.)
—— & Bader, M. N., 888 (Typh.)
——, with ——, 639 (Typh.)
Ann. Trop. Med. & Parasit., 780 (Mal.)

Annecke, A., McCarthy, D. D. & Wilson, D. B. 966 (Mal.)
Antes, E. H., with Paul & Sahs, 804 (Dengue)
Antunes, W. S., with Soper, Wilson & Lima, 79 (B.R.)
Apley, J. & Grant, G. H., 836 (Misc. Dis.)
Applebaum, I. L. & Shrager, J., 176 (Mal.)
—— & Wexberg, L. E., 816 (Hel.)
Arana, R. & Asenjo, A., 908 (Hel.)
Arbona, G., 990 (Typh.)
Arbuse, D. I., 963 (Mal.)
d'Arcangelo, D., 1002 *bis* (Am.)
Argüelles Casals, D., 214, 568 (Lep.)
Armbrust, C. A., with Kohls, Irons & Philip, 993 (Typh.)
Armytage, J. E. & Bolliger, A., 358 (Leish.)
Arnaud, R., with Sarrouy, Dendale, Combe & Gillot, 102 (Leish.)
Aschenbrenner, R., 25, 104 (Typh.)
—— & Marx, R., 105 (Typh.)
Ascher, L., (143) (Der.)
Asenjo, A., with Arana, 908 (Hel.)
Ashe, W. F., with Eichna, Bean & Nelson, 751 (Heat Str.)
Ashley, J. D., Jr., with Halpert, 126 (Am.)
Ashton, D. F., with Schoof & Schell, 626 (Mal.)
de Assumpção, L. 277 (Y.F.)
Atwell, A. G. & Bailey, N., 209 (Am.)
Auerbach, M. E., with Barlow & Rivenburg, 443 (Mal.)
—— & Eckert, H. W., 90 (Mal.)
Augustine, D. L., Weinman, D. & McAllister, J., 288 (R.F.)
Augustson, G. F., (281) (Pl.)
——, with Pollard, 551 (Typh.)
Austin, C. J., 294, 736 (Lep.)
Austin, T., 290 (Lep.)
Australian Military Forces, 63 (Vms.)
Austrian, R., with Whitehill, 470 *bis* (Ys.)
d'Avanzo, C. S., 1026 (Der.)
Avtsyn, A. P., 266 (Typh.)
Axtmayer, J. H., with Krakower & Hoffman, 133 (Hel.)

B.

Back, R. C., with Huffaker, 687 (Mal.)
——, with Rice & Huffaker, 632 (Mal.)
Bader, M. & Anigstein, L., 639 (Typh.)
——, with ——, 888 (Typh.)
——, with Blair, 799 (Typh.)
Baer, L. S., & Allen, R. R., 419 (Reports, etc.)
Baert, H., with Bessemans, Derom, Doussy & Steyaert, 870 (Tryp.)
Bagrova, A. S., with Gromashevsky & Stepanov, 460 (Typh.)
Bailey, N., with Atwell, 209 (Am.)
Bailly, J., with Remlinger, 169, 170 (Rab.)
Baker, D. H., 76 (Reports, etc.)
Bakhrami, D. A., 454 (Leish.)
Balagangadharan, K. V., 739 (Lep.)
Balamuth, W. & Sandza, J. G., 999 (Am.)
Balasquide, L. A. (6) (Mal.)
Ball, A. L., with Gordon, Lippincott, Marble, Ellerbrook & Glass, 692 (Mal.)
Ball, D. & Ryan, R. C., 19 (Leish.)
Ball, E. G., Anfinsen, C. B., Geiman, Q. M., McKee, R. W. & Ormsbee, R. A., 867 (Mal.)
Bally, P. R. O., 658 (Misc. Dis.)
Baltazard, M., with Blanc, 33, 376 (Pl.), 713 *bis*, 714, 715 *bis*, (719) (Typh.)
——, with —— & Martin, 713 (Typh.)
Banerjee, P. K., with Das Gupta & Sen Gupta, 129 (Am.)
Barber, M. A., (229) (Ent.)
Bardhan, P. N., 29 (Typh.)
Barlovatz, A., with Rodhain, 887 (Typh.)
Barlow, C. H. & Abdel Azim, M., 1017 (Hel.)
Barlow, O. W., Auerbach, M. E. & Rivenburg, H., 443 (Mal.)
Barnard, J. H., with Sprague, 890 (Y.F.)
Barnes, R. C., (627) (Mal.)
Barnett, L., 399, (742) (Hel.)
Barr, D. P., with Harrison & Tompsett, 222 (Sp.)
Barreto, M. P., 185 (Mal.)
—— & Coutinho, J. O., (150) (Ent.)
Barrett, N. R. & Thomas, D., 474 (Hel.)
Barrow, M., 506 (Reports, etc.)
Bartter, F. C., 649 (Hel.)
Basombrio, G., with Mom, 1012 (Lep.)
Basu, B. C. & Sen, S., 645 (R.F.)
Basu, U. P., with Sen, 893 (Chl.)
Bates, M., 72 (Ent.)
Bates, R. D., Jr., with Coggeshall & Martin, 698 (Mal.)
Battelli, C., Coccani, A. & Rossi, M., 977 (Leish.)
Bauer, C. R., with Maveety & Turnbull, 441 (Mal.)
Bauer, J. & Fisher, L. J., 484 (Haem.)
Baumann, E., 844 (Lab.)
Baumann, H., with Schwetz & Fort, 810 (R.F.)
Bax, S. N., 702 (Tryp.)
Baylis, H. A., 302, 906 (Hel.)
Bayliss, M., with Swantz, 701 (Bl.)
Bayoumi, A., with Kirk, 374 (Y.F.)
Beach, H. L. W. & Rennie, J. K., 880 (Typh.)
Bean, W. B., with Eichna, Ashe & Nelson, 751 (Heat Str.)
Bearcroft, W. G. C., with van Rooyen, Darskin & Pollack, 194 (Typh.)
Bebey, M., with Martin, 37 (Am.)
Bechelli, L. M., Keil, H. & Rotberg, A., 900 (Lep.)
Beckman, H., 433 (Mal.)
Beet, E. A., 856 (Mal.)
Begg, A. M., Fulton, F. & van den Ende, M., 104 (Typh.)
Begg, F. H. H., with Anderson & McNaughton, 497 (Misc. Dis.)
Begg, R. C., 46 (Hel.)
Beguet, M., with Durand, Horrenberger & Renoux, 107 (Typh.)
——, with Sergent, 270 (Typh.)
Beiglböck, W. & Löscher, H., 31 (Typh.)
Beklemishev, V. N. & Gontaeva, A. A., 248 (Mal.)
Belferman, S., with Shapiro & Saliternik, 247 (Mal.)
Bell, E. J., with Blake, Maxcy, Sadusk & Kohls, 991, 993 (Typh.)
Bell, F. R., with Carmichael, 259, 260 (Tryp.)
Bell, S. D., Jr. & Brown, H. W., 820 (Hel.)
Bellows, J. G., 489 (Oph.)
Beltrán, E., 361 (Leish.), 594 (Prot.), 895 (Am.)

Beltrán, E., with Guerra, De la Garza & Raúl Larenas, 866 (Mal.)
—— & Raúl Larenas, M., 20 (Leish.), 36 (Am.), 594 (Prot.)
Benesch, R., with Ellinger, 926 (Def. Dis.)
Bengtson, I. A., 983 (Typh.)
——, with Brigham, 552 (Typh.)
Benians, T. H. C., 95 (Bl.)
Bennett, B., with Plotz, Wertman & Snyder, 636 (Typh.)
Bennison, B. E. & Coatney, G. R., 623 (Mal.)
Berberian, D. A., 102, 359, 360 *bis*, 877 (Leish.)
Bercovitz, Z. T., with Oliver-González, 219 (Hel.)
——, Rodriguez-Molina, R., Hargrave, D. W., Dickie, J. D. & Green, C. E., 47 (Hel.)
——, Shwachman, H. & Rodriguez-Molina, R., 572 (Hel.)
Berger, E. & Brzezinski, S., 986 (Typh.)
Berk, J. E., 592 (Misc. Dis.)
Berkowitz, A. P., 107 (Typh.)
Berliner, R. W., with Shannon, Earle, Brodie, Taggart *et al.*, 343 (Mal.)
Berman, C., 321 (Misc. Dis.)
Bernard, M., with Poursines, Pigoury & Borde, (189) (Tryp.)
Bernard, P. N. & Gallut, J., 34 *bis*, 807 (Chl.)
Bernasconi, E. M., 488 *bis* (Oph.)
Berry, J. N., (784) (Mal.)
Berry, M. G., Johnson, A. S., Jr. & Warshauer, S. E., 555 (Typh.)
Berteaux, M., with Wanson, 337 (Mal.)
Bertho, A., 39 (Am.)
Bessemans, A. & Derom, R., with Doussy, P., Baert, H. & Steyaert, J., 870 (Tryp.)
de Bettencourt, A., with de Oliveira, 166 (Rab.)
Bevier, G., 557 (Y.F.), 614 (Mal.)
Bhaduri, N. V., with Mukerji, 919 (Hel.)
Bhattacharjya, B. P., 935 (Misc. Dis.)
Bhide, B. V., with Paranjape, Phalnikar & Nargund, 571 (Hel.)
Bieler, R., 204 (Typh.)
Bieloglazov, G. G., 249 (Mal.)
Biggs, R., with Sharp, 1042 (Misc. Dis.)
Bilibin, 796 (Typh.)
Binckley, E. C., with Manwell, Coulston & Jones, 839 (Prot.)
Bingham, J. A. W. & McDonald, S., 305 (Hel.)
Binns, R. T., 835 (Misc. Dis.)
Bird, J., 686 (Mal.)
Bird, W., (253) (Mal.)
Birnbaum, D., Goldblum, N. & Kligler, I. I., 700 (Bl.)
Bischoff, G., 198 (Typh.)
Bishop, E. L. & Gartrell, F. E., 184 (Mal.)
Bishopp, F. C., (661) (Ent.)
—— & Trembley, H. L., 760 (Ent.)
Bispham, W. N., 945 (B.R.)
Black, D. A. K., 494 (Heat Str.)
Black, L. O'C., with de Meillon & Parent, (940) (Ent.)
Black-Schaffer, B., with Levy, 833 (Der.)
Blackie, W. K., (95) (Bl.)
Blacklock, D. B., 91, 255 (Mal.), 326, 506 (Reports, etc.)
Blagoveshchenskiĭ, D. I., Bregetova, N. G. & Monchadskiĭ, A. S., 230 (Ent.)
Blair, R., with Glasgow, 4 (Mal.)
Blair, R. K. & Bader, M. N., 799 (Typh.)
Blake, F. G., Maxcy, K. F., Sadusk, J. F., Jr., Kohls, G. M. & Bell, E. J., 991, 993 (Typh.)
Blanc, G. & Baltazard, M., 33, 376 (Pl.), 713 *bis*, 714, 715 *bis*, (719) (Typh.)
——, Martin, L. A. & Baltazard, M., 713 (Typh.)
—— & Woodward, T. E., 547 (Typh.)
Bland, E. F., with Woodward, 199 (Typh.)
Bland, J. O. W., 1034 (Oph.)
—— & Wilson, R. P., 1031 (Oph.)
Bloom, H., 121 (Dys.)
Bloss, J. F. E., 969 (Tryp.)
——, with Hunt, 969 (Tryp.)
Boase, A. J., 1033 (Oph.)
Bocharova, T. V., 274 (Typh.)
Bogen, E., 861 (Mal.)
Böger, A., 130 (R.F.)
Bohart, G. E., with Knight & Bohart, 511 (B.R.)
Bohart, R. M., with Farner, 503 (Ent.)
——, with Knight & Bohart, 511 (B.R.)
Bohls, S. W., with Irons, Thurman & McGregor, 462 (Typh.)
Bol. Oficina Sanitaria Panamericana, 87 (Mal.)
Bolliger, A., with Armytage, 358 (Leish.)
Bomford, R. R., 384 (Am.)
Bone, G., with van den Berghe, (843) (Ent.)
Bonfiglioli, H., with Negroni, 586 (Der.)
Bonnard, A., 140 (Def. Dis.)
Boquet, P., 309, (584), 652, 1026 (Vms.)
——, with Ramon, Richou, Nicol & Delaunay-Ramon, (584) (Vms.)
Borde, R., with Poursines, Pigoury & Bernard, (189) (Tryp.)
v. Bormann, F., 462 (Typh.)
Bose, A. N. & Ghosh, J. K., 207 (Dys.)
——, —— & Rakshit, P. C., 444 (Mal.), 468 (Am.)
—— & Rakshit, P., 451 (Mal.)
Bose, B. C. & Ahuja, H. L., 121 (Chl.)
Bose, N., with Kirwan & Sen, 1036 (Oph.)
Boshell-Manrique, J. & Osorno-Mesa, E., 114 (Y.F.)
Boshoff, P. H., 1036 (Oph.)
Bourgain, M., with Pirot, 137 (Hel.), 811 (R.F.)
Bourguignon, G. C., 172 (Mal.)
Bowie, J. H., with van Rooyen & Krikorian, 264 (Typh.)
Boyd, M. F., 175 (Mal.)
—— & Kitchen, S. F., 174 (Mal.)
Bozicevich, J., with Rees, Reardon, & Daft, 36 (Am.)
Bracey, P., with David & Harvey, 229 (Ent.)
Bradley, G. H., (229) (Ent.), 446 (Mal.)
Brady, F. J., Lawton, A. H., Cowie, D. B., Andrews, H. L., Ness A. T. & Ogden, G. E., 747 (Hel.)
Brahmachari, U., 178 (Mal.)
Brambell, F. W. R. & Davis, D. H. S., 281 *bis* (Pl.)
Branday, W. J., with McFarlane, 755 (Misc. Dis.)
Brandenburger, P., 26 (Typh.)
Braun, H. & Unat, E. K., 108 (Typh.)
Braun, K., with Rachmilewitz, 821 (Def. Dis.)
—— & de Vries, A., (351) (Mal.)

Brauss, F. W., 355 (Tryp.)
Brazzaville [A.E.F.], 710 (Tryp.), 802 (Y.F.), 995 (Typh.)
Breaks, V., with Alicata, 271 (Typh.)
Breault, H. J., 676 (Rab.)
Bregetova, N. G., with Blagoveshchenskiĭ & Monchadskiĭ, 230 (Ent.)
Brennan, J. M., 799 (Typh.)
Briand, L. J., with Monro, Delisle & Smith, 759 (Ent.)
Briggs, A. P., Singal, S. A. & Sydenstricker, V. P., 821 (Def. Dis.)
Brigham, G. D. & Bengtson, I. A., 552 (Typh.)
British Empire Leprosy Relief Association, 290 (Lep.)
British Guiana, 557 (Y.F.), 614 (Mal.), 737 (Lep.)
British Medical Journal, 88 (Mal.)
Brock, J. F., 308 (Haem.)
Brodie, B. B., with Shannon, Earle, Taggart, Berliner *et al.*, 343 (Mal.)
Bromberg, L., with Byrd & St. Amant, 479 (Hel.)
Bronson, L. H., Jr., with Chamberlain, 1035 (Oph.)
Bronstein, L. H., with Potter & Gruber, 690 (Mal.)
Brooke, M. M., 864 (Mal.)
Brooks, A. G., with Ahuja, 830 (Vms.)
——, with Malone, 357 (Leish.)
Broom, J. C., with Alston, 210 (R.F.)
Browaeys, J., 189 (Tryp.)
Brown, A., 1022 (Haem.)
—— & Anderson, A. B., 1023 (Haem.)
Brown, E. H., (1044) (Ent.)
Brown, G. M., with Maegraith, Rossiter, Irvine, Lees, Parsons, Partington, Rennie & Havard, 251 (Mal.)
Brown, H. W., 53 (Hel.)
—— with Bell, 820 (Hel.)
Brown, J. H., 805 (Pl.)
Brown, J. S., 779 (Mal.)
—— & Trowell, H. C., 306 (Def. Dis.)
Brown, M. & Rennie, J. L., 780 (Mal.)
Brown, R. L., 644 (Am.)
Browne, D. C., McHardy, G. & Spellberg, M.A., 729 (Am.)
Browning, C. H., Browning, P. & Robb, J. V. M., 258 (Tryp.)
—— & Calver, K. M., 259 (Tryp.)
——, —— & Leckie, M. W., 709 (Tryp.)
Browning, P., with Browning & Robb, 258 (Tryp.)
Brozek, J., with Henschel, Taylor, Mickelsen & Keys, 145 (Heat Str.)
Brugsch, H. G. & Gill, D., 141 (Haem.)
Brumpt, E., (967) (Mal.)
Brumpt, L., 197 (Typh.)
Brumpt, L.-C., with Duvoir, 913 (Hel.)
Brunt, D., 928 (Heat Str.)
Brzezinski, S., with Berger, 986 (Typh.)
Btesh, S., 128 (Am.)
Bull. Agric. Congo Belge, (849) (Reports, etc.)
Bull. Inst. Hyg. Maroc., (233 *bis*), (234) (Reports, etc.)
Bull. U. S. Army Med. Dept., 659 (Ent.), 788 (Bl.), 1019 (Hel.)
Bulmer, E., 288 (R.F.)
Burch, G. E., 752 (Heat Str.)
—— & Winsor, T., 752 *bis* (Heat Str.)
——, with ——, 484, 1024 (Haem.), 752 (Heat Str.)
Burch, J. C., with Quill, 225 (Der.)
Burchenal, J. H. & Woods, R. P., 635 (Leish.)
Burgess, P., 298 (Lep.)
Burgess, R. W., with Eylés, (627) (Mal.)
—— & Young, M. D., 437 (Mal.)
Burke, 405 (Ulcer)
Burke, E., 232 (Reports, etc.)
—— & Chakravarty, K. C., 17 (Leish.)
Burroughs, A. L. & Holdenried, R., 387 (R.F.)
Burrows, W., 727 (Chl.)
——, Mather, A. N., Wagner, S. M. & McGann, V. G., 727 (Chl.)
——, Wagner, S. M. & Mather, A. N., 728 (Chl.)
Burruss, H. W., with Hargett, 803 (Y.F.)
Burtt, E., 868 (Tryp.)
Burtt, E. D., (356) (Tryp.)
Bushland, R. C., Eddy, G. W. & Knipling, E. F., 660 (Ent.)
——, with Mariotte & Varela, 721 (Typh.)
Bustamante, M. E. & Varela, G., 203 (Typh.)
Busvine, J. R., 151, 839 (Ent.)
Butt, H. R., Hall, J. E., Watkins, C. H. & Cragg, R. W., 783 (Mal.)
——, with Walters, Watkins & Marshall, 37 (Am.)
Buxton, P. A., 335 (Mal.), 839 (Ent.)
Byrd, D. A., with Yolles & Yolles, 437 (Mal.)
Byrd, E. E., St. Amant, L. S. & Bromberg, L., 479 (Hel.)
Byrne, E. A. J., with Croxon, J. H., 142 (Der.)
Byron, R. A., with Lipman, Casey & Evans, 371 (Typh.)

C.

Cairo, 1017 (Hel.)
Caldwell, F. E., 94 (Mal.)
Calewaert, D., 12 (Bl.)
Callahan, W. P., Jr., 486 (Der.)
Callot, J. & Dao Van Ty, (594) (Ent.)
Calver, K. M., 704 (Tryp.)
——, with Browning, 259 (Tryp.)
——, with —— & Leckie, 709 (Tryp.)
Cambournac, F. J. C., (1043) (Ent.)
Cameron, T. W. M., (153) (Ent.), 510 (Reports, etc.)
Campbell, G. A. & West, T. F., 152 (Ent.)
Canaan, T., 894 (Am.)
Cantrell, W. & Jordan, H. B., 633 (Mal.)
Carboni, E., with Fernández, Vaccaro & Serial, 832 (Der.)
Cardis, F., 1039 (Misc. Dis.)
Cardoso, H. T., with da Cunha, Guimarães & Leão, 1003 (Ys.)
——, with ——, Leão & Guimarães, 1003 (Ys.)
Carley, P. S., (822) (Def. Dis.)
Carmichael, J., 979 (Typh.)
—— & Bell, F. R., 259, 260 (Tryp.)
Carmody, J. T. B., with McGinn, 341 (Mal.)
Carothers, J. C., 658 (Misc. Dis.)

Carpanelli, J. B., with Latienda, 137 (Hel.)
Carpenter, S. J. (229) (Ent.)
Carr, D. T. & Essex, H. E., 889 (Bart.)
Carragher, A. E., 288 (R.F.)
Carri, E. L., 731 (Am.)
Carter, H. F., Wedd, G. & d'Abrera, V. St. E., 73 (Ent.)
Case, R. A. M., 1023 (Haem.)
Casey, A. V., with Lipman, Byron & Evans, 371 (Typh.)
Castañeda, M. R., 27, 716 (Typh.)
—— & Silva, G. R., 193, 457 (Typh.)
——, with ——, 990 (Typh.)
Castro G., J., 573 (Hel.)
Castro Estrada, S., with Varela & Olarte, 927 (Der.)
——, with Zozaya & Varela, 143 (Der.)
de Castro Palomino, J., 44 (Lep.)
——, with Grau Triana & Conde Mateo, 213 (Lep.)
Catanei, A., with Montpellier, 67 (Der.)
——, with Parrot & Collignon, 445 (Mal.)
Cathie, I. A. B., (595) (Reports, etc.)
Causey, O. R., Deane, M. P., da Costa, O. & Deane, L. M., 647 (Hel.)
Cavazzi, G., 113 (Typh.)
Cawston, F. G., 815 (Hel.)
Cayer, D., Ruffin J. M. & Perlzweig, W. A., 580 (Def. Dis.), 1021 (Sp.)
Ceccaldi, J., 710 (Tryp.), 802 (Y.F.), 995 (Typh.)
Cecil, R. C., with Scherer, 583 (Haem.)
Ceithaml, J., with Silverman, Taliaferro & Evans, 448 (Mal.)
Celestin, W., with Kahn & Offenhauser, 661 (Ent.)
Cenac, P. L., with Michael & McLaughlin, 66 (Der.)
Cerqueira, F. M. C., with Corrêa, (72) (Ent.)
Cerruti, H., 567 (Lep.)
Chabaud, A., with Chorine, 298 (Lep.)
Chabeuf, M., 936 (Misc. Dis.)
Chagas, A. W., (876) (Leish.)
Chakravarti, H., with Das Gupta & Siddons, 177 (Mal.)
Chakravarty, K. C., with Burke, 17 (Leish.)
Chamberlain, W. P., Jr. & Bronson, L. H., Jr., 1035 (Oph.)
Chambers, H. D. 131 (Ys.)
Chambers, W. E., with Hammick (349) (Mal.)
Chandler, A. C., 32 (Y.F.) 757 (Ent.)
——, with Addis, 302 (Hel.)
Chang, K., 759 (Ent.)
—— & Ch'in, H. T., 744 (Hel.)
——, with Tang, 743 (Hel.)
——, Tong, W. K., Chin, H. T. & Li, C. H., 744, (Hel.)
Chang, K. P., with Chu & Kuo (927) (Def. Dis.)
Chang S. L., with Fair, Taylor & Wineman, 734 (Am.)
Chang, S. S., with Shih & Yuan, 726 (Pl.)
Chargaff, E., with Cohen, 192 (Typh.)
Charters, A. D., 813 (Ys.)
Chatterjee, B. C., with Paul, 379 (Chl.)
Chatterjee, M. L., with Pal, Mukerji & Gupta, 286 (Am.)
Chatterji, S. N., with Dharmendra, (901) (Lep.)
Chaudhuri, R. N. & Rai Chaudhuri, M. N., 221 (Sp.), (838) (Misc. Dis.), 962 (Mal.)
Chen, G. & Geiling, E. M. K., 348 (Mal.)
——, —— & MacHatton, R. M., 869 *bis*, 872 (Tryp.)
Chen, S. C., with Kao, 756 (Misc. Dis.)
Chen, Y. P., Zia, S. H. & Anderson, H. H., 734 (R.F.)
Cheney, G., with Denenhölz, 485 (Der.)
Cheng, W. S., Liu, Y. P. & Tan, S. C., 733 (Am.)
Chesterman, C. C., 10 (Bl.)
Chhuttani, P. N., with Malhotra, 59 (Haem.)
——, with Taylor, 750 (Haem.)
Chi, C. Y., with Wu, 795 (Typh.)
Ch'in, H. T., with Chang, 744 (Hel.)
——, with ——, Tong & Li, 744 (Hel.)
Chin, Y. C., with Chou, 70 (Misc. Dis.)
Ch'ing Hua Meng & Winfield, G. F., (842 *bis*) (Ent.)
Chon, A., with Sohier & Parnet, (795) (Typh.)
Chorine, V., 298, 1013 (Lep.)
—— & Chabaud, A., 298 (Lep.)
—— & Colas-Belcour, J., (811) (R.F.)
Chorley, T. W., 98 (Tryp.)
Chou, C. & Chin, Y. C., 70 (Misc. Dis.)
Chow, H. K., with Chung, (876) (Leish.)
Chu, C. M., with Tang & Wong, 120 (Chl.)
Chu, H. I., Kuo, P. T. & Chang, K. P., (927) (Def. Dis.)
Chuan, T. T. K., with Anderson, 563 (Am.)
Chung, H. L., 876 (Leish.)
—— & Chow, H. K., (876) (Leish.)
——, Wang, C. W. & Lee, C. U., (876) (Leish.)
Chwatt, L. J., 860 (Mal.)
——, with Gilroy, 786 (Mal.)
Cilli, V. & Corazzi, G., 827 (Vms.)
Cimmino, V., 102, 106 (Typh.)
Clavero, G. & Olavarria, J., (859) (Mal.)
—— & Pérez Gallardo, F., 794 (Typh.)
Clearkin, P. A., 326 (Reports, etc.), 497 (Misc. Dis.), 915 (Hel.)
Clough, F. E., 751 (Der.)
Clow, J. M., 101 (Leish.)
Coatney, G. R., with Bennison, 623 (Mal.)
—— & Cooper, W. C., 353 (Mal.)
——, —— & Miles, V. I., 865 (Mal.)
——, —— & Trembley, H. L., 865 (Mal.)
Coceani, A., with Battelli & Rossi, 977 (Leish.)
Cochran, D. M., 62 (Vms.)
Cochrane, E., 86 *bis*, (Mal.)
Cochrane, R. G., 291 (Lep.)
Cocker. D. E., 79 (B.R.)
Coggeshall, L. T., (577) (Hel.)
——, Martin, W. B. & Bates, R. D., Jr., 698 (Mal.)
——, Porter, R. J. & Laird, R. L., 699 (Mal.)
Coghill, N. F., 1001 (Am.)
Cogswell, R. C., with Spies & Viltner, 307 (Def. Dis.)
Cohen, A. G., 564 (R.F.)
Cohen, H. G., (130) (R.F.)
Cohen, L. & Harrison, I., 643 (Am.)
Cohen, S. S., 981 (Typh.)
—— & Chargaff, E., 192 (Typh.)
Colah, R. B. M., with Rao & Kalle, 932 (Ulc.)

Colas-Belcour, J., with Chorine, (811) (R.F.)
Cole, C. L., with Hawkins, 912 (Hel.)
Coleman, M. B., 250 (Mal.)
Collier, J. M., (85) (Mal.)
Collignon, (171) (Mal.)
Collignon, E., 698 (Mal.)
——, with Parrot & Catanei, 445 (Mal.)
Collingwood, F. C., 255 (Mal.), 282 (Chl.)
Collins, J. D., 117 (Den.)
Colonial Office, 721 (Y.F.), 760 (Misc.)
Combe, P., with Sarrouy, Dendale, Arnaud & Gillot, 102 (Leish.)
Comerford, C. & Kirman, B. H., (822) (Def. Dis.)
Conde Mateo, E., with Grau Triana & de Castro Palomino, 213 (Lep.)
Connell, W. K., 228 (Ulc.)
Consolazio, F. C., with Pitts, Johnson, Poulin, Razoyk & Stachelek, 226 (Heat Str.)
Cook, A. B., 220 (Sp.)
Cook, C. E., 367 (Typh.)
Cooley, R. A. & Kohls, G. M., 154 (B.R.)
Cooper, T. & Wessels, A. L., 857 (Mal.)
Cooper, W. C., with Coatney, 353 (Mal.)
——, with —— & Miles, 865 (Mal.)
——, with —— & Trembley, 865 (Mal.)
Cooray, G. H., 149 (Misc. Dis.)
Copping, A. M., 749 (Def. Dis.)
Corazzi, G., with Cilli, 827 (Vms.)
Cornatzer, W. E., with Andrews, 534 *bis* (Mal.)
Corrêa, R. R. & Cerqueira, F. M. C., (72) (Ent.)
——, with Galvão & Grieco, 4 (Mal.)
—— & Ramos, A. S. (173), 961 (Mal.)
Cort, W. W., 905 (Hel.)
Cossio, F., with Romaña, 874 (Tryp.)
da Costa, O., with Causey, Deane & Deane, 647 (Hel.)
Costa, O. G., 151 (Ent.), 264, 544 (Leish.)
Cot Lesmes, V., 44 (Lep.)
Couceiro, A., 975 (Tryp.)
Coulston, F., with Huff, 538 (Mal.)
——, with Manwell, Binckley & Jones, 839 (Prot.)
——, with Taliaferro & Silverman, 449 (Mal.)
Coutinho, B., with Magalhães, Gouvêa, Lucena & Ignacio, 1016 (Hel.)
Coutinho, J. O., with Barreto, (150) (Ent.)
Cova-Garcia, P., 250 (Mal.)
Covell, G., 623 (Mal.)
Cowie, D. B., with Brady, Lawton, Andrews, Ness & Ogden, 747 (Hel.)
Cragg, R. W., with Butt, Hall & Watkins, 783 (Mal.)
Cram, E. B. & Hicks, D. O., 818 (Hel.)
——, Jones, M. F. & Wright, W. H., 646 (Hel.)
Cross, R. M., 469 (R. F.)
Croxon, J. H., with Byrne, 142 (Der.)
Cruickshank, J. C., with Nelson, 363 (Typh.)
Cruz, W. O. & de Mello, R. P., 1020 (Hel.)
Culbertson, J. T. & Rose, H. M., 136 (Hel.)
Culpepper, G. H., 594 (Ent.)
da Cunha, A. M., Guimarães, F. N., Leão, A. E. A. & Cardoso, H. T., 1003 (Ys.)
——, Leão, A. E. A., Guimarães, F. N. & Cardoso, H. T., 1003 (Ys.)
Cupi, N., 132 (Ys.)

D.

Daft, F. S., with Rees, Bozicevich, & Reardon, 36 (Am.)
Daggy, R. H., 32 (Den.)
Dähne, G., 338 (Mal.)
Dakin, W. P. H., with Harrison, 176 (Mal.)
Dammin, G. J. & Weller, T. H., 941 (Lab.)
Damon, S. R. & Johnson, M. B., 637, 990 (Typh.)
Danskin, D., with van Rooyen, 463 (Typh.)
Dao, L. L., 697 (Mal.), 17, 711 (Tryp.)
Dao Van Ty, with Callot, (594) (Ent.)
Darskin, D., with van Rooyen, Pollack & Bearcroft, 194 (Typh.)
Dartevelle, E., with Schwetz, (815 *bis*), (1017) (Hel.)
Darzins, E., 544 (Typh.)
Das Gupta, B. M., 427 (B.R.)
—— & Ganguli, S. K., 338 (Mal.)
—— & Siddons, L. B., 537 (Mal.)
——, —— & Chakravarti, H., 177 (Mal.)
Das Gupta, C. R., with Majumder (141) (Haem.)
Das Gupta, N. C., Banerjee, P. K. & Sen Gupta, S. B., 129 (Am.)
Davey, T. F., 1013 (Lep.)
Davey, T. H., with Gordon, 541 (Bl.)
David, W. A. L., 499 (Ent.)
——, Bracey, P. & Harvey, A., 229 (Ent.)
Davidson, L. R., (476) (Hel.)
Davidson, S., 964 (Mal.)
Davies, A. B., with Williams, (404) (Vms.)
Davis, D. E., 758 (Ent.)
Davis, D. H. S., with Brambell, 281 *bis* (Pl.)
Davis, L. J., 595 (Ent.)
Davison, A. R., 292, 293 *bis*, 295 (Lep.)
De, S. S., 224 (Vms.)
Deane, L. M., with Causey, Deane & da Costa, 647 (Hel.)
Deane, M. P., with Causey, da Costa & Deane, 647 (Hel.)
Deckert, K., with Hauer, 809 (Am.)
Degotte, J., 295, 394 (Lep.)
De la Camara, J. P., 984 (Typh.)
De la Garza, F., with Guerra, Beltrán & Raúl Larenas, 866 (Mal.)
Delaunay-Ramon, Mme., with Ramon, Boquet, Richou & Nicol, (584) (Vms.)
Delgado, E., with Hurtado & Merino, 824 (Haem.)
Delisle, R., with Monro, Briand & Smith, 759 (Ent.)
Delperdange, G., 741 (Lep.)
Del Pozo, E. C., Anguiano, L. G., & González, Q. J., 653 (Vms.)
Del Sel, M., (999) (Am.)
DelVecho, S. B., with MacMahon, 506 (Lab.)
Dembovitz, N., 508 (Reports, etc.)
Demoulin, M. L. & Parent, M., 845 (Lab.)
——, with ——, (688) (Mal.)
Dendale, R., with Sarrouy, Combe, Arnaud & Gillot, 102 (Leish.)
Denenholz, E. J. & Cheney, G., 485 (Der.)
Denhoff, E., with Hodge & Vander Veer, 1020 (Hel.)
Densham, W., 392 (Lep.)
Derom, R., with Bessemans, Doussy, Baert & Steyaert, 870 (Tryp.)

von Deschwanden, J., with Anderson, Gray & Menzies, 961 (Mal.)
Desportes, C., (1020) (Hel.)
Devignat, R., (727), 806 (Pl.)
Dharmendra & Chatterji, S. N., (901) (Lep.)
—— & Mukherji, N., 900, 903 (Lep.)
—— & Rajagopalan, S., 1012 (Lep.)
—— & Santra, I., 738 (Lep.)
Dias, C. B., with Magalhães, 1016 (Hel.)
Dias, E., with Romana, 974 (Tryp.)
—— & Torrico M., R.A., (875) (Tryp.)
Diasio, J. S. & Richardson, F. MacD., 205 (Den.)
Dick, G. W. A., 1038 (Misc. Dis.)
Dickie, J. D., with Bercovitz, Rodriguez-Molina, Hargrave & Green, 47 (Hel.)
Dillon, R. A., (327) (Reports, etc.)
Ding, E., 22 (Typh.)
Diniz, O., 933 (Ulc.)
Diwani, M., 224 (Haem.)
Dixon, H. B. F. & Hargreaves, W. H., 907 (Hel.)
Dixon, K. C. & Fuller, W., 469 (R.F.)
Dju, M. Y., with Hou, 650, (927) (Def. Dis.)
Doak, G. O., with Eagle, Hogan & Steinman, 15 (Tryp.)
Dolman, C. E., with Ranta, 467 (Chl.)
Domenjoz, R., 500 (Ent.)
Dominguez López, F., (44) (Lep.)
Donin, L., with Segura, Radice & Giusti, (141) (Haem.)
Donovick, R. & Wyckoff, R. W. G., 792, 882 (Typh.)
Dos Santos Neves, E., 472 (Lep.)
Doussy, P., with Bessemans, Derom, Baert & Steyaert, 870 (Tryp.)
Dove, W. E., 502 (Ent.)
——, with Knipling, 660 (Ent.)
Dreisbach, R. H. & Hanzlik, P. J., 964 (Mal.)
Drew, R., with Hodge, 559 (Am.)
Drew, W. R. M. & Reid, J., 862 (Mal.)
Drewe, F. S., 292 (Lep.)
Drobyshevskaya, A. I., with Smorodintsev, 196 (Typh.)
Dubois, A., 835 (Misc. Dis.)
Dugard, J. H., 416 (Reports, etc.)
Duke, H. L., 13 (Tryp.)
Dulaney, A. D. & Morrison, D. B., 352 (Mal.)
Durán, C. M., 458 (Typh.)
Durand, P., Beguet, M., Horrenberger, R. & Renoux, G., 107 (Typh.)
Durieux, C., 275, 723 (Y.F.), 289 (R.F.), (326) (Reports, etc.), 678 (Rab.)
Duvoir, M. & Brumpt, L. C., 913 (Hel.)
Duvolon, S., with Stéfanopoulo, 205 (Y.F.)
van Dyke, H. B., with Stauber, 540 (Mal.)

E.

Eagle, H., 354 (Tryp.)
——, Hogan, R. B., Doak, G. O. & Steinman, H. G., 15 (Tryp.)
—— & Magnuson, H. J., 356 (Tryp.)
Earle, D. P., Jr., with Shannon, Brodie, Taggart, Berliner *et al.*, 343 (Mal.)
Earle, K. V., 64 (Vms.), 927 (Def. Dis.), 1041 (Misc. Dis.)
East African Med. J., 251 (Mal.), 659, 754 (Misc. Dis.)
Eberlin, E., 31 (Typh.)
Eccles, C. G. & Ross, H. 297 (Lep.)
Ecke, R. S., with Yeomans, Snyder, Murray & Zarafonetis, 201 (Typh.)
Eckert, H. W., with Auerbach, 90 (Mal.)
Eckstein, A., 860 (Mal.)
Eddey, L. G., 255 (Mal.)
Eddy, G. W., with Bushland & Knipling, 660 (Ent.)
Edmunds, P. K., 464 (Typh.)
Edney, E. B., 504 (Ent.)
Ehrman, F. C., Ellis, J. M. & Young, M. D., 628 (Mal.)
Eichna, L. W., Bean, W. B., Ashe, W. F. & Nelson, N., 751 (Heat Str.)
Einhorn, N. H., with Miller, 578 (Hel.)
——, ——, & Whittier, L., 911, 912, 914 (Hel.)
Elek, S. R., 374 (Den.)
Elford, W. J. & van den Ende, M., 455 (Typh.)
Elkeles, G., 872 (Tryp.)
Elkerton, L. E., 543 (Leish.)
Elkinton, J. R. & Winkler, A. W., 316 (Heat Str.)
Ellerbrook, L. D., with Gordon, Lippincott, Marble, Ball & Glass, 692 (Mal.)
Ellinger, P. & Benesch, R., 926 (Def. Dis.)
Elliott, M., 276 (Y.F.)
Ellis, J. M., with Ehrman & Young, 628 (Mal.)
Ellis, M. D., with Ellis & Westfall, 256 (Mal.)
Ellis, M. M., Westfall, B. A. & Ellis, M. D., 256 (Mal.)
Emerson, G. A., with Siegel & Mushett, 628 (Mal.)
Emmons, C. W., 833 (Der.)
v. Engel, R., 49 (Hel.)
Engelberg, R., with Pirk, 964 (Mal.)
Englehorn, T. D. & Wellman, W. E., 819 (Hel.)
Engley, F. B., Jr., with Morton, 997 (Dys.)
Enikolopov, S. K., 859 (Mal.)
Ephraim, H., (66) (Der.)
Epidemiological Information Bull. (UNNRA Health Division), Wash., 724 (Pl.), 890 (Y.F.)
Essex, H. E., (584) (Vms.)
——, with Carr, 889 (Bart.)
Etévé, J., with Stéfanopoulo, 258 (Tryp.)
Etherington, D., 173 (Mal.)
Etteldorf, J. N., with Harned, 443 (Mal.)
Evans, E. A., Jr., with Silverman, Ceithaml & Taliaferro, 448 (Mal.)
——, with Speck, 967 (Mal.)
Evans, E. C., with Lipman, Casey & Byron, 371 (Typh.)
Evans, H. M., 937 (Misc. Dis.)
Ewing, F. M., with Haas, 539 (Mal.)
——, with —— & Feldman, 865 (Mal.)
Ewing, H. E., (231), 505 (Ent.)
Eyer, H. & Ruska, H., 23 (Typh.)
Eyles, D. E., 435 (Mal.)
—— & Burgess, R. W., (627) (Mal.)

F.

Faget, G. H., with Hopkins, 737 (Lep.)
—— & Pogge, R. C., 297, 646 (Lep.)
Fahey, J., with Petersen, 920 (Hel.)

Faierstein, S. G., 456 (Typh.)
Failmezger, T. R., with Jacoby, Loudon & Wyne, 559 (Dys.)
Fain, A., 14 (Tryp.)
Fair, G. M., Chang, S. L., Taylor, M. P. & Wineman, M. A., 734 (Am.)
Fairbairn, D., (584) (Vms.)
Fairbairn, H., 452 (Tryp.)
Fairley, N. H., 630 (Mal.), 656 (Misc. Dis.)
Falcão, P. C., 935 (Misc. Dis.)
Familiari, P., 490 (Oph.)
Fan, J. H., 1048 (Reports, etc.)
Fanta, H., with Siedek & Kasperczik, 105 (Typh.)
Farinaud, M. E., (1021) (Hel.)
Farner, D. S., 503 (Ent.)
—— & Bohart, R. M., 503 (Ent.)
—— & Katsampes, C. P., 272 (Typh.)
——, with Knight, 688 (Mal.)
Farooq, M. & Qutubuddin, M., 978 (Leish.)
Fasal, P., 138 (Def. Dis.), 831 (Der.)
Fassi, F., with Gaud & Salm, 420 (Reports, etc.)
Faust, E. C., 154 (Reports, etc.), 319 (Misc. Dis.)
——, Scott, J. A. & McDaniel, G. E., 620 (Mal.)
Feder, I. A., 231 (Ent.)
Fehily, L., 139 (Def. Dis.)
Feinman, M. H., 228 (Ulc.)
Feldman, H. A., with Haas & Ewing, 865 (Mal.)
Feldman-Muhsam, B., 413 (Ent.)
Felix, A., 979 (Typh.)
Felsen, J., 379 (Dys.)
Felsenfeld, O., 283 (Chl.)
Feo, L. G., 72 (Prot.)
Fernandez, J. M. M., 297, 471 (Lep.)
——, Vaccaro, A., Serial, A. & Carboni, E., 832 (Der.)
Fernández Castanys, A., 978 (Leish.)
Ferreira, F. da C. & Ferreira, T., (1044) (Ent.)
Ferreira, T., with Ferreira, (1044) (Ent.)
Ferreira, T. G., with de Meira, (1044) (Ent.)
Ferriman, D. G., 438 (Mal.)
—— & Mackenzie, G. K., 124 (Dys.)
Ferris, A. A., with Fortune, 808 (Dys.)
Ferro-Luzzi, G., 67 (Misc. Dis.), 100 (Leish.), 478 (Hel.)
Fetterman, G. H., Moran, T. J. & Hess, W. R., 495 (Misc. Dis.)
Fielding, J. W., 903 (Lep.)
Fiennes, R. N. T. W., Jones, E. R. & Laws, S. G., 791 (Tryp.)
Fife, J. G., 680 (Rab.)
de Figueiredo, F. V., 222 (Sp.)
Filatov, V. P., 491 (Oph.)
Findlay, G. M., Hill, K. R. & Macpherson, A., 289 (Ys.)
——, with Holden, 503 (Ent.)
——, with Maegraith, 12 (Bl.)
——, Markson, J. L. & Holden, J. R., 6, 350 (Mal.)
—— & Stevenson, A. C., 349 (Mal.)
Finerman, W. B. & Weiss, J. E., 284 (Dys.)
Finlay, G. M., 630 (Mal.)
Finnegan, J. D., with Measham & Yoeli, 695 (Mal.)
Finnerud, C. W., 308 (Def. Dis.)
Fischbach, W. M. & Howell, D. E., (995) (Typh.)
Fisher, G. E., 57 (Def. Dis.)
Fisher, L. J., with Bauer, 484 (Haem.)
Fisher, R. A., with Hertig, 758 (Ent.)
Fishman, J., 312 (Der.)
Fitz-Hugh, T., Jr., with Gaskill, 629 (Mal.)
——, Pepper, D. S. & Hopkins, H. U., 340 (Mal.)
Fitzpatrick, F. K., 793, 982, 996 (Typh.)
Fleck, E. E., with Hall & Schechter, 842 (Ent.)
Flecker, H., 68, (937) (Misc. Dis.), (225) (Vms.)
—— & McSweeny, A., 66 (Der.)
Fleming, A. McK., 48 (Hel.)
Flood, C. A. & Sherman, W., 419 (Reports, etc.)
Florio, L., Stewart, M. O. & Mugrage, E. R., 118 (Den.)
——, with —— & ——, (119) (Den.)
Fogel, R. H. & Huntington, R. W., Jr., 53 (Hel.)
Forrester, J. S., with Machella, 994 (Typh.)
Fort, M., with Schwetz & Baumann, 810 (R.F.)
Fortune, C. & Ferris, A. A., 808 (Dys.)
Foster, V. W., (466) (Pl.)
Fowler, A. F., 541 (Tryp.)
Fox, L. A., 979 (Typh.)
Foy, H., Kondi, A., Rebelo, A. & Soeiro, A., 634 (Bl.)
Francisco, R., 642 (Den.)
Francisco Ruiz R., 748 (Hel.)
Franks, M. B. & Stoll, N. R., 916 (Hel.)
Fraser, J. F. & Taylor, R., 897 (Am.)
Freeborn, S. B., (229) (Ent.)
Freed, L. F., (594) (Prot.)
Freis, E. D. & Mater, D. A., 402 (Sp.)
de Freitas, G., with Muniz, 975 (Tryp.)
French, M. H., (943) (Reports, etc.)
Frith, D., with Hammick, 8 (Mal.)
Fromme, W. & Gaase, A., 197 (Typh.)
Frost, K., with Obermayer, 832 (Der.)
Fuller, A. T., 264 (Leish.), 1048 (Lab.)
Fuller, W., with Dixon, 469 (R.F.)
Fulton, F., 979 (Typh.)
——, with Begg & van den Ende, 104 (Typh.)
Fulton, J. D., 19, 978 (Leish.)

G.

Gaase, A., 481 (Hel.)
——, with Fromme, 197 (Typh.)
Gage, D. P., with Thomas, 1018 (Hel.)
Gajewski, J. E. & Tatum, A. L., 92 (Mal.)
Galindo, P., with Mangabeira, (151) (Ent.)
Gallardo, E., Sanz, J. & Perez Gallardo, F., 23 (Typh.)
Gallut, J., with Bernard, 34 *bis*, 807 (Chl.)
—— & Grabar, P., 807 (Chl.)
Galvão, A. L. A., Corrêa, R. R. & Grieco, S. J., 4 (Mal.)
Ganguli, S. K., with Das Gupta, 338 (Mal.)
Ganguly, S. C., with Gupta, 943 (Reports, etc.)
Garnham, P. C. C., 959 (Mal.)
—— & Harper, J. O., 447 (Mal.)

Garratt, E. I., 649 (Hel.)
Gartrell, F. E., with Bishop, 184 (Mal.)
Garvan, J. M., with Wall, 893 (Dys.)
Gaschen, H., 512 (B.R.)
Gaskill, H. S. & Fitz-Hugh, T., Jr., 629 (Mal.)
Gasperini, G. C. (42) (R.F.)
Gaud, J., Salm, G. & Fassi, F., 420 (Reports, etc.)
Gayid, I. K., 5 (Mal.)
Gaynor, J. S., with Russ, (533) (Mal.)
Gear, H. S., (1045) (Ent.)
Gear, J., 362 (Typh.)
——, with Wolstenholme, 877 (Typh.)
Geigy, R., 684 (Mal.)
Geiling, E. M. K., with Chen, 348 (Mal.)
——, with —— & MacHatton, 869 *bis*, 872 (Tryp.)
Geiman, Q. M., with Ball, Anfinsen, McKee & Ormsbee, 867 (Mal.)
Gelfand, M., 78 (B.R.), 148 (Misc. Dis.), 943 (Reports, etc.)
Geller, W., 9 (Mal.)
Georgetown, British Guiana, 326 (Reports, etc.)
Gerberg, E. J., with Gordon, (940) (Ent.)
German, W. M., 819 (Hel.)
——, with McGavack, 141 (Haem.)
Getting, V. A., (662) (Ent.)
Ghalioungui, P., 57 (Def. Dis.)
Ghosh, J. K., with Bose, 207 (Dys.)
——, with —— & Rakshit, 444 (Mal.), 468 (Am.)
Ghosh, T. K. (65) (Vms.)
di Giacomo, M. P. & Mayer, R. A., 47 (Hel.)
Gibson, T. E., 396 (Hel.)
Giffen, H. K., 906 (Hel.)
Giglioli, G., 615 (Mal.)
Gilbert, W., 490 (Oph.)
Gilbertson, W. E., 641 (Den.)
Gilchrist, M., with King, 781, 782 (Mal.)
Gilford, G. H., 487 (Oph.)
Gill, C. A., 282 (Chl.)
Gill, D., with Brugsch, 141 (Haem.)
Gillman, J., with Gillman, 748 (Def. Dis.), 1046 (Lab.)
Gillman, T. & Gillman, J., 748 (Def. Dis.), 1046 (Lab.)
Gillot, F., with Sarrouy, 101 (Leish.)
——, with ——, Dendale, Combe & Arnaud, 102 (Leish.)
Gilroy, A. B. & Chwatt, L. J., 786 (Mal.)
Ginsburg, J. M., 939 (Ent.)
Gintscheff, P. Z., 882 (Typh.)
Giordano, A. F., 37 (Am.)
Girard, G., 375, 377 (Pl.)
Giroud, P., 199, 366 *bis*, (Typh.)
—— & Panthier, R., 104, 985 (Typh.)
Giusti, C. L., with Segura, Radice & Donin (141) (Haem.)
Gjullin, C. M., with Knipling & Yates, 73 (Ent.)
Glasgow, R. D. & Blair, R., 4 (Mal.)
Glass, W. W., Jr., with Gordon, Lippincott, Marble, Ball & Ellerbrook, 692 (Mal.)
Glauser, F., 479 (Hel.)
Gloeckner, A., with Kessel, Allison, Kaime & Quiros, 40 (Am.)
Góbich, E., with Rivas & Mantilla, 910 *bis* (Hel.)
van Goidensenhoven, C., with Rodhain, 869 (Tryp.)
—— & Schoenaers, F., 791 (Tryp.)
Golberg, L., de Meillon, B. & Lavoipierre, M., (1043) (Ent.)
——, with —— & —— (1043) (Ent.)
Gold, E. M., with Wright, 1041 (Misc. Dis.)
Goldblum, N., with Birnbaum & Kligler, 700 (Bl.)
Golden, A., 718 (Typh.)
Goldman, L., 480 (Hel.), 843 (Ent.)
Gontaeva, A. A., with Beklemishev, 248 (Mal.)
González, Q. J., with Del Pozo & Anguiano, 653 (Vms.)
González Ochoa, A. & Ruiloba, J., 142 (Der.)
Gonzalez Peris, G., with Trespalacios & de Laosa, 586 (Der.)
Gonzalez Prendes, M. A., with Ibarra Perez, (214), 1009 (Lep.)
González Ureña, J., (1010) (Lep.)
Goobar, J. K., with Savino, 33, 466 (Pl.)
Good, R., 91 (Mal.)
Goodwin, L. G., 262 (Leish.)
Goodwin, M., with Mulrennan & Shannon, 633 (Mal.)
Gordon, H. H., Lippincott, S. W., Marble, A., Ball, A. L., Ellerbrook, L. D. & Glass, W. W., Jr., 692 (Mal.)
Gordon, R. M., 255 (Mal.)
—— & Davey, T. H., 541 (Bl.)
——, with Hill, 940 (Ent.)
Gordon, W. M. & Gerberg, E. J., (940) (Ent.)
Goriacheva, L. K., 575 (Hel.)
Gorsse, P. & Accart, R., 395 (Hel.)
Gotshalk, H. C., (1041) (Misc. Dis.)
Gottfried, S. P., 885 (Typh.)
Gouck, H. K., with Smith, 151 (Ent.)
Gouvêa, L., with Magalhães, Coutinho & Ignacio, 1016 (Hel.)
Grabar, P., with Gallut, 807 (Chl.)
Graham, J. G. & Naftalin, J. M., 876 (Leish.)
Di Grandi, J., with von Sallmann & Meyer, 489 (Oph.)
Grant, G. H., with Apley, 836 (Misc. Dis.)
Grant, J., Anderson, M. & Thompson, R. B., 187 (Tryp.)
Grasset, E., 829 (Vms.)
Grau, M. L., 232 (Lab.)
Grau Triana, J., 212 (Lep.)
——, de Castro Palomino, J. & Conde Mateo, E., 213 (Lep.)
Gray, W. A., with Anderson, von Deschwanden & Menzies, 961 (Mal.)
Green, C. E., with Bercovitz, Rodriguez-Molina, Hargrave & Dickie, 47 (Hel.)
Green, F. H. K., with Hawking, 1052 (B.R.)
Green, R. A., 442 (Mal.)
Greenburg, N., with Heilman & Knutson, 898 *bis* (R.F.)
Greenslade, C. M. & Morrison, D. (229) (Prot.)
Greiff, D., Pinkerton, H. & Moragues, V., 362 (Typh.)
Grieco, S. J., with Galvão & Corrêa, 4 (Mal.)
Griffitts, J. J., 284 (Chl.)
Grindley, D. N., with Henry, 783 (Mal.)
Gromashevsky, L. V., Bagrova, A. S. & Stepanov, I. R., 460 (Typh.)
—— & Stepanov, I. R., 191 (Typh.)

Grosfilez & Lefèvre, (1038) (Misc. Dis.)
Gruber, C. M., with Potter & Bronstein, 690 (Mal.)
Guasch, J., 426 (B.R.)
Guerra, F., Beltrán, E., De la Garza, F. & Raúl Larenas, M., 866 (Mal.)
Guggenheim, K., with Kligler & Schwartz, 930 (Heat Str.)
Guhl, R., 51 (Hel.)
Guimarães, F. N., 812 (Ys.)
——, with da Cunha, Leão & Cardoso, 1003 *bis* (Ys.)
Gulamova, V. P., with Vainberg, Perekriostova & Ostrovskaya, 549 (Typh.)
Gunther, C. E. M., 849 (B.R.)
Gupta, J. C., & Ganguly, S. C., 943 (Reports, etc.)
—— & Kahali, B. S., 191 (Leish.)
——, with Pal, Mukerji & Chatterjee, 286 (Am.)
Gupta, L. M., with Ransome & Paterson, 146 (Misc. Dis.)
Gupta, R. D. & Laha, P. N., 87 (Mal.)
Gutmann, M. J., 1031 (Oph.)
Gutsevich, A. V., 533 (Mal.)
Guyer, O. K. G., with Manson-Bahr, 409 (Misc. Dis.)

H.

Haas, E., 252 (Mal.)
Haas, V. H. & Ewing, F. M. 539 (Mal.)
——, Feldman, H. A. & Ewing, F. M., 865 (Mal.)
Hackett, C. J., 255 (Mal.)
Hadaway, A. B., with Symes, 785 (Mal.)
Haddow, A. J., with Smithburn, 320 (Misc. Dis.)
——, with —— & Mahaffy, 321 (Misc. Dis.)
Haine, G. L., with Skipper, 451 (Bl.)
Halbeisen, W. A., with Stryker & Leventhal, 581 (Def. Dis.)
Hall, J. E., with Butt, Watkins & Cragg, 783 (Mal.)
Hall, S. A., Schechter, M. S. & Fleck, E. E., 842 (Ent.)
Hall, T. F., with Hess, 626 (Mal.)
Hallervorden, J., 638 (Typh.)
Halpert, B. & Ashley, J. D., Jr., 126 (Am.)
Hamilton, H. L., Plotz, H. & Smadel, J. E., 796 (Typh.)
Hamilton, T. R., with Angevine, Wallace & Hazard, 976 (Leish.)
Hamm, W. G. & Ouary, G., 495 (Ulc.)
Hammick, D. L. & Chambers, W. E., (349) (Mal.)
—— & Frith, D., 8 (Mal.)
Hammond, A G., 1038 (Ulc.)
Hamre, D., with Jones & Rake, 543 (Leish.)
Hanschell, H. M., 845 (Reports, etc.)
Hanzlik, P. J., with Dreisbach, 964 (Mal.)
Hardenbergh, W. A., (229) (Ent.)
Hardesty, J. F., 1036 (Oph.)
Harding, R. D. & Hawking, F., 188 (Tryp.)
Hardwick, S. W., 926 (Def. Dis.)
Hardy, J. D. & Passmore, R., (977) (Leish.)
Hargett, M. V., 558 (Y.F.)
—— & Burruss, H. W., 803 (Y.F.)
Hargrave, D. W., with Bercovitz, Rodriguez-Molina, Dickie & Green, 47 (Hel.)
Hargreaves, W. H., 559, 895 (Am.)
——, with Dixon, 907 (Hel.)
Harington, C. & Young, E. A. E., 271 (Typh.)
Harned, B. K. & Etteldorf, J. N., 443 (Mal.)
Harper, A. R., (1027) (Der.)
Harper, J. G. M., with Almy, 646, 815 (Hel.)
Harper, J. O., with Garnham, 447 (Mal.)
Harrell, G. T. & Horne, S. F., (926) (Hel.)
——, with ——, 306 (Hel.)
——, Venning, W. & Wolff, W. A., 373 (Typh.)
——, Wolff, W. A. & Venning, W., 888 (Typh.)
Harris, P. N., 595 (Reports, etc.)
Harrison, H. E., Tompsett, R. R. & Barr, D. P., 222 (Sp.)
Harrison, I., with Cohen, 643 (Am.)
Harrison, K. S. & Dakin, W. P. H., 176 (Mal.)
Hart, V. L., 211 (R.F.)
Hartz, H., with van der Sar, 837 (Misc. Dis.)
Hartz, P. H., 480 (Hel.), 592 (Misc. Dis.)
Harvey, A., with David & Bracey, 229 (Ent.)
Harvey, A. M., 963 (Mal.)
Hasche-Klünder, R., 167 (Rab.)
Hatch, W. E. & Wells, A. H., 312 (Der.)
Hauer, A. & Deckert, K., 809 (Am.)
Havard, R. E., with Maegraith, 12 (Bl.), 863 (Mal.)
——, with ——, Brown, Rossiter, Irvine, Lees, Parsons, Partington & Rennie, 251 (Mal.)
——, with —— & Parsons, 968 (Bl.)
Hawking, F., 536 (Mal.), 971 (Tryp.)
—— & Green, F. H. K., 1052 (B.R.)
——, with Harding, 188 (Tryp.)
Hawkins, P. A., & Cole, C. L., 912 (Hel.)
Hay, C. P., 109 (Typh.)
Hayman, J. M., Jr., 252 (Mal.)
Hazard, J. B., with Angevine, Hamilton & Wallace, 976 (Leish.)
Headlee, T. J., 763 (B.R.)
Heaslip, W. G., with Womersley, 28 (Typh.), 324 (Ent.)
Hecht, O., 281 (Pl.), 323 (Ent.)
—— & Anduze, P. J., 465, 640 (Y.F.)
Hegsted, D. M., McKibbin, J. M. & Stare, F. J., 784 (Mal.)
Heidelberger, M. & Mayer, M. M., 689 (Mal.)
Heilig, R. & Naidu, V. R., 30 (Typh.)
—— & Visveswar, S. K., 127 (Am.), 442 (Mal.)
Heilman, F. R., with Knutson, M. & Greenburg, N., 898 *bis* (R.F.)
Heisch, R. B., 658 (Misc. Dis.)
Helander, E. V., 512 (B.R.)
Helander, S., 415 (Lab.)
Held, A., 113 (Typh.)
Helfet, A. J., 387 (Ys.)
Heller, E. R., 579 (Hel.)
Helme, A. C. de B., 10 (Bl.)
Hemming, F., 244 (Mal.)
Hemming, G. R., 897 (Am.)
Henderson, A. B., with West, 253 (Mal.)
Henderson, C. A., 374 (Y.F.)
Hennessey, R. S. F., 730 (Am.)
Henrard, C., van Wymeersch, H. & Wanson, M., (229) (Ent.)
——, with van Hoof & Peel, 96 (Tryp.)

Henry, A. J. & Grindley, D. N., 783 (Mal.)
——, with Kirk, 18 (Leish.)
Henschel, A., Taylor, H. L., Brozek, J., Mickelsen, O. & Keys, A., 145 (Heat Str.)
Herrera, J. R., 265 (Typh.)
Hertel, H. W., 496 (Misc. Dis.)
Hertig, M. & Fisher, R. A., 758 (Ent.)
Herzog, E., 676 (Rab.)
Hess, A. D. & Hall, T. F., 626 (Mal.)
—— & Kiker, C. C., 183 (Mal.)
——, with Kruse & Metcalf, 184 (Mal.)
——, with Metcalf, 351 (Mal.)
——, with Rozeboom, 182 (Mal.)
Hess, W. R., with Fetterman & Moran, 495 (Misc. Dis.)
Heyden, G., 491 (Oph.)
Hiatt, E. P., 178 (Mal.)
—— & Quinn, G. P., 778 (Mal.)
Hibbard, J. S., with Zuckerman, 480 (Hel.)
Hicks, D. O., with Cram, 818 (Hel.)
Hicks, J. D., 554 (Typh.)
Hill, K. R., with Findlay & Macpherson, 289 (Ys.)
Hill, M. A. & Gordon, R. M., 940 (Ent.)
Hindle, J. A., Rose, A. S., Trevett, L. D. & Prout, C., 446 (Mal.)
Hirst, W. R. & McCann, W. J., 838 (Misc. Dis.)
Hirt, G., 682, 683 (Rab.)
Hoare, C. A., 559 (Am.)
Hoare, E. D., 794 (Typh.)
Hochstein, E., with Kellner & Tillman, (964) (Mal.)
Hodes, P. J. & Keefer, G. P., 848 (Reports, etc.)
Hodge, E. H. V., 630 (Mal.)
—— & Drew, R., 559 (Am.)
Hodge, I. G., Denhoff, E. & Vander Veer, J. B., 1020 (Hel.)
Hofer v. Lobenstein, M., (106) (Typh.)
Hoffa, E., with Sorsby, 488 (Oph.)
Hoffman, W. A., with Krakower & Axtmayer, 133 (Hel.)
Hogan, R. B., with Eagle, Doak & Steinman, 15 (Tryp.)
Hogue, M. J., 228 (Prot.)
Holden, J. R. & Findlay, G. M., 503 (Ent.)
——, with —— & Markson, 6, 350 (Mal.)
——, with Saunders & Hughes, 354 (Tryp.)
Holdenried, R., with Burroughs, 387 (R.F.)
Hole, N., 979 (Typh.)
v. Hollósy, K., (219) (Hel.)
Hoodless, D. W., 416 (Reports, etc.)
van Hoof, L., Henrard, C. & Peel, E., 96 (Tryp.)
van Hoof, M. T., with Rodhain, (778) (Mal.), (791) (Tryp.)
Hopkins, G. H. E., 230 (Ent.)
Hopkins, H. U., with Fitz-Hugh & Pepper, 340 (Mal.)
Hopkins, R. & Faget, G. H., 737 (Lep.)
Horne, S. F. & Harrell, G. T., 306 (Hel.)
——, with ——, (926) (Hel.)
Horrenberger, R., with Durand, Beguet & Renoux, 107 (Typh.)
—— & Renoux, G., 107 (Typh.)
——, with Sergent, 365 (Typh.)
Hou, H. C., 651 (Def. Dis.)
—— & Dju, M. Y., 650, (927) (Def. Dis.)
Hou, P. C., (100) (Leish.)
Howard, A. C., 10 (Bl.)
Howat, H. T., 123 (Dys.)
Howell, D. E., with Fischbach, (995) (Typh.)
Howorth, M. B., 817 (Hel.)
Hsu, K. C., (41) (Am.)
Hsue, L. T., 726 (Pl.)
Hu, S. M. K., 918 (Hel.)
Hudson, E. H., (863) (Mal.)
Hudson, J. R., 678 (Rab.)
Hudson, M. F., with Ladell & Waterlow, 143 (Heat Str.)
Huff, C. G. & Coulston, F., 538 (Mal.)
Huffaker, C. B. & Back, R. C., 687 (Mal.)
——, with Rice & Back, 632 (Mal.)
Hughes, K. T. & Niesche, F. W., (628) (Mal.)
Hughes, M. H., with Saunders & Holden, 354 (Tryp.)
Hühne, W., 447 (Mal.)
Hume, W. E. & Szekely, P., 210 (R.F.)
Hundley, J. M. & Nasi, K. W., 282 (Pl.)
Hunt, A. R., 1019 (Hel.)
—— & Bloss, J. F. E., 969 (Tryp.)
Hunt, R. S., (810) (Am.)
Hunt, T. C., 5 (Mal.)
Hunt, V. L., 766 (B.R.)
Hunter, G. W., III, with Mackie & Worth, 665 (B.R.)
Huntington, R. W., Jr., with Fogel, 53 (Hel.)
Hurst, H., (941) (Ent.)
Hurtado, A., Merino, C. & Delgado, E., 824 (Haem.)
Hussain, N., 881 (Typh.)
Hyman, A. S., 962 (Mal.)
Hynes, M., 822 (Haem.)
——, Ishaq, M. & Morris, T. L., 651 (Haem.)

I.

Ibarra, R., (44) (Lep.)
Ibarra Perez, R. & Gonzalez Prendes, M. A., (214), 1009 (Lep.)
Ibbotson, C., with Menon, 884 (Typh.)
Ignacio, L., with Magalhães, Coutinho, Gouvêa & Lucena, 1016 (Hel.)
Ignacio Chala, H. J. & Lleras Restrepo, F., 566 (Lep.)
Imrie, A. H., 169 (Rab.)
India, 406 (Misc. Dis.), 822 (Haem.)
Indian Med. Gaz., 30 (Typh.), 283 (Chl.)
Inhelder, H. E., (862) (Mal.)
Iriarte, D. R., 655 (Der.)
Irons, E. N., with Kohls, Armbrust & Philip, 993 (Typh.)
Irons, J. V., Bohls, S. W., Thurman, D. C., Jr. & McGregor, T., 462 (Typh.)
Irvine, C., 406 (Ulc.)
Irvine, K. N., with Maegraith, Brown, Rossiter, Lees, Parsons, Partington, Rennie & Havard, 251 (Mal.)
Isaicheva, A. I., with Podyapolskaya, 573 (Hel.)
Isbister, J., 749 (Sp.)
Ishaq, M., with Hynes & Morris, 651 (Haem.)
Isola, W. & Osimani, J. J. (230) (Ent.)
Iswariah, V., (394) (Lep.)
Iyengar, M. O. T., 625 (Mal.)

J.

Jachowski, L. A., with Wilson & Matheison, 73 (Ent.)
Jack, R. W., (14) (Tryp.)
Jackson, A. V., 244 (Mal.)
——, with Williams & Sinclair, 369 (Typh.)
Jackson, C. H. N., 186 (Tryp.)
—— & Vanderplank, F. L., 187 (Tryp.)
Jackson, D. S., 881 (Typh.)
Jacob, J. E., with Tomlinson, 582 (Haem.)
Jacob, V. P., with Jaswant Singh, 618 (Mal.)
Jacobs, H. R., 465 (Y.F.)
Jacobs, L., with Wright & Walton, 306 (Hel.)
Jacoby, A. H., Loudon, J. R., Wyne, P. S. & Failmezger, T. R., 559 (Dys.)
Jaffe, H. L., 918 (Hel.)
James, S. P., 255, 630 (Mal.), 282 (Chl.), 845 (Reports, etc.)
Jang, C. S., 630 (Mal.)
Jansen, G., 1018 (Hel.)
——, with Nery Guimarães, 972 (Tryp.)
——, with Pondé & Mangabeira, (876) (Leish.)
Jarvis, J. F., 932 (Ulc.)
Jaswant Singh & Jacob, V. P., 618 (Mal.)
Jayawardena, M. D. S., with Soysa, 320 (Misc. Dis.)
Jeffery, G. M., 352 (Mal.)
Jellison, 405 (Ulc.)
Jellison, W. L., 805 (Pl.)
Jennings, R. K., 806 (Chl.)
—— & Linton, R. W., 378 *bis*. (Chl.)
——, with ——, 377 (Chl.)
Jettmar, H. M. & Shigan, Li, 981 (Typh.)
Johnson, A. S., Jr., with Berry & Warshauer, 555 (Typh.)
Johnson, M. B., with Damon, 637, 990 (Typh.)
Johnson, P. A. G., 478 (Hel.)
Johnson, R. E., with Pitts, Consolazio, Poulin, Razoyk & Stachelek, 226 (Heat Str.)
Johnstone, D. F., 489 (Oph.)
Jokl, E., 317 *bis* (Heat Str.)
de Jolinière, P. B., with Parrot, (662) (Ent.)
Jones, E. R., with Fiennes & Laws, 791 (Tryp.)
Jones, H., Rake, G. & Hamre, D., 543 (Leish.)
Jones, M. F., with Cram & Wright, 646 (Hel.)
Jones, S. H. & Souders, C. R., 149 (Misc. Dis.)
Jones, V. P., with Manwell, Coulston & Binckley, 839 (Prot.)
Jonescu, D., 674, 677 (Rab.)
Jordan, H. B., with Cantrell, 633 (Mal.)
Jörg, M. E., with Mazza & Miyara, 792 (Tryp.)
Josephs, H. W., 61 (Haem.)
J. Amer. Med. Ass., 799 (Typh.)
J. Indian Med. Ass., 936 (Misc. Dis.)
Jover, J., with Trespalacios, 212 (Lep.)
Jukova, N. N., 940 (Ent.)
Juon, M., 389 (Lep.)

K.

Kahali, B. S., with Gupta, J. C., 191 (Leish.)
Kahn, M. C., Celestin, W. & Offenhauser, W., 661 (Ent.)
Kaime, M., with Kessel, Allison, Quiros & Gloeckner, 40 (Am.)
Kalle, R. A., with Rao & Colah, 932 (Ulc.)
Kammer, V., 48 (Hel.)
Kanchaveli, G. I., 632 (Mal.)
Kane, F. F., 42 (R.F.)
Kanibolotskaya, E. N., with Steinberg & Karlova, 339 (Mal.)
Kanof, A., with Sulzberger & Shaw, 1028 (Der.)
Kao, Y. E. & Chen, S. C., 756 (Misc. Dis.)
Kaplan, A. & Lindgren, A., 996 (Den.)
Kaplan, D., 735 (R.F.)
Karlova, N. S., with Steinberg & Kanibolotskaya, 339 (Mal.)
Kartagener, M., with Spühler, 1039 (Misc. Dis.)
Kasimov, B., 50 (Hel.)
Kasperczik, K., with Siedek & Fanta, 105 (Typh.)
Kassirsky, I. A., 628 (Mal.)
Katsampes, C. P., with Farner, 272 (Typh.)
Katzenellenbogen, I., 361 (Leish.)
Katzenstein, L., 899 (Ys.)
Kauntze, W. H., 255 (Mal.)
Kay, C. F., 696 (Mal.)
Kean, B. H., 359 (Leish.), 420 (Reports, etc.)
—— & Smith, J. A., 338 (Mal.)
Keefer, G. P., with Hodes, 848 (Reports, etc.)
Keegan, H. L., with Olson, (504) (Ent.)
Keh-Wei-Huang, 69 (Misc. Dis.)
Keil, H., with Bechelli & Rotberg, 900 (Lep.)
Keilin, D., 150 (Ent.)
Kellner, A., Hochstein, E. & Tillman, A. J. B. (964) (Mal.)
Kennaway, E. L., 150 (Misc. Dis.)
Keon-Cohen, B. T., with Lawes, (234) (Reports, etc.)
Kern, R. A. & Norris, R. F., 341 (Mal.)
Kessel, J. F., Allison, D. K., Kaime, M., Quiros, M. & Gloeckner, A., 40 (Am.)
Kessler, W. R. & Zwemer, R. L., 889 (Bart.)
Keys, A., with Henschel, Taylor, Brozek & Mickelsen, 145 (Heat Str.)
Khan, H., (92) (Mal.)
Khan, N. U., 861 (Mal.)
Khokhlova-Buyanova, O. F., 230 (Ent.)
Khodukin, N. I., 356 (Leish.)
Khramushin, J. E., with Pshenichnov, 324 *bis* (Ent.)
Kiaer, W., with Petri & Nørgaard, (308) (Def. Dis.)
Kiang, L., with Kuo, 741 (Hel.)
Kibler, M., 639 (Typh.)
Kiker, C. C., with Hess, 183 (Mal.)
Kikuth, W., 102 (Leish.)
King, B. G., 304 (Hel.)
King, E. J. & Gilchrist, M., 781, 782 (Mal.)
King, G., 845 (Reports, etc.)
King, H., 971 (Tryp.)
——, with Andrewes, van den Ende & Walker, 20 (Typh.)
Kirby, H., 838 (Prot.)
Kirk, R., 190, 875 (Leish.)
—— & Bayoumi, A., 374 (Y.F.)
—— & Henry, A. J., 18 (Leish.)
Kirkaldy-Willis, W. H., 740 (Lep.)
Kirman, B. H., with Comerford (822) (Def. Dis.)
Kirwan, E. O'G., Sen, K. & Bose, N., 1036 (Oph.)
Kitchen, S. F., with Boyd, 174 (Mal.)

Kleinhaus, E. M., with Lyon, 1040 (Misc. Dis.)
Kleinman, A., Page, R. C. & Preisler, P. W., (829) (Vms.)
Kligler, I. I., with Birnbaum & Goldblum, 700 (Bl.)
Kligler, I. J., Guggenheim, K. & Schwartz, I., 930 (Heat Str.)
—— & Oleinik, E., 23 (Typh.)
Klitzner, H., 455 (Leish.)
Klopstock, A. & Steinitz, H., 590 (Misc. Dis.)
Knight, K. L., Bohart, R. M. & Bohart, G. E., 511 (B.R.)
—— & Farner, D. S., 688 (Mal.)
Knipe, F. W., with Russell, Ramachandra Rao & Patnam, 687 (Mal.)
Knipling, E. F., with Bushland & Eddy, 660 (Ent.)
—— & Dove, W. E., 660 (Ent.)
——, Gjullin, C. M. & Yates, W. W., 73 (Ent.)
——, with Lindquist & Schroeder, 1044 (Ent.)
——, with Madden & Lindquist, 73 (Ent.)
Knott, J. I., 497 (Misc. Dis.)
Knutson, M., with Heilman & Greenburg, 898 *bis* (R.F.)
Koepfli, J. B., with Mead, 9 (Mal.)
Kohls, G. M., Armbrust, C. A., Irons, E. N. & Philip, C. B., 993 (Typh.)
——, with Blake, Maxcy, Sadusk & Bell, 991, 993 (Typh.)
——, with Cooley, 154 (B.R.)
Kojevnikov, P. V., 876 (Leish.)
Kojevnikova, E. V., 454 (Leish.)
Kondi, A., with Foy, Rebelo & Soeiro, 634 (Bl.)
Korshunova, O. S., 274 (Typh.)
——, with Skorin, 638 (Typh.)
Kossovaja, E. M., with Alisov, 989 (Typh.)
Kouri, P., 477 (Hel.)
Kovács, G. & v. Kup, J., 461 (Typh.)
Krakower, C., Hoffman, W. A. & Axtmayer, J. H., 133 (Hel.)
Kremer, H., with Ruge, 85 (Mal.)
Krick, E. S., with Vonfraenkel, 592 (Misc. Dis.)
Krikorian, K. S., with van Rooyen & Bowie, 264 (Typh.)
Krontovskaya, M. K. & Shmatikov, M. D., 274 (Typh.)
Kruse, C. W., Hess, A. D. & Metcalf, R. L., 184 (Mal.)
Kubes, V., 422 (B.R.), 454 (Tryp.)
Kudo, R. R., 327 (B.R.)
Kuhn, M. J., with Savino & Morales Villazon, 558 (Pl.)
Kunstadter, R. H. & Prendergrass, R. C., (655) (Der.)
Kuo, P. T., with Chu & Chang, (927) (Def. Dis.)
Kuo, S. & Kiang, L., 741 (Hel.)
v. Kup, J., with Kovács, 461 (Typh.)

L.

Ladell, W. S. S., Waterlow, J. C. & Hudson, M. F., 143 (Heat Str.)
Laender, J. F., with Lopes, 979 (Leish.)
Lagos, Nigeria, 800 (Y.F.)
Laha, P. N., 936 (Misc. Dis.)
——, with Gupta, 87 (Mal.)
Lahiri, S. C., 643 (Chl.)
Laird, R. L., with Coggeshall & Porter, 699 (Mal.)
Lal, H. B., 590 (Misc. Dis.)
Lam, C. R., with McClure, 627 (Mal.)
Lamb, W. L. & Royston, G. R., 561 (Am.)
Lampert, H., 365 (Typh.).
Lancet, 88, 342, 779 (Mal.), 680 (Rab.)
Lane, C., 400 (Hel.)
Langer, K., 76 (Reports, etc.)
Länger, P., Martin, H. & Muller, P., 499 (Ent.)
de Laosa, O., with Trespalacios & Gonzalez Peris, 586 (Der.)
Lapping, 405 (Ulc.)
Larsh, J. E., Jr., 322 (Prot.)
Laser, H., (219) (Hel.)
Laskin, 795 (Typh.)
Latienda, R. I. & Carpanelli, J. B., 137 (Hel.)
Laure, G., 267 (Typh.)
Lavoipierre, M., with Golberg & de Meillon, (1043) (Ent.)
——, with de Meillon & Golberg, (1043) (Ent.)
Lawes, C. H. W. & Keon-Cohen, B. T., (234) (Reports, etc.)
Laws, S. G., with Fiennes & Jones, 791 (Tryp.)
Lawton, A. H., with Brady, Cowie, Andrews, Ness & Ogden, 747 (Hel.)
Leão, A. E. A., with da Cunha, Guimarães & Cardoso, 1003 *bis* (Ys.)
Leckie, M. W., with Browning & Calver, 709 (Tryp.)
Ledent, H., 75 (Reports, etc.)
Ledentu, (172) (Mal.)
Lee, C. U., with Chung & Wang, (876) (Leish.)
Lee, D. H. K., 76 (Reports, etc.)
Lee, D. J. & Woodhill, A. R., 1049 (B.R.)
Lee, R. K. C. & Pang, H. Q., 593 (Misc. Dis.)
Lee, R. V., 313 (Der.)
Lees, J. C., with Maegraith, Brown, Rossiter, Irvine, Parsons, Partington, Rennie & Havard, 251 (Mal.)
Lefèvre with Grosfilez, (1038) (Misc. Dis.)
LeFevre, I. D., Jr., McDermott, K. F. & Venner, R. B., 470 (Ys.)
Leffkowitz, M. & Sukiennik, S., 591 (Misc. Dis.)
Le Gall, R., (215) (Hel.), (233) (Reports, etc.), 280 (Pl.)
Leitman, M. Z., 254 (Mal.)
Lemaire, G., 268 (Typh.)
Lennette, E. H. & Perlowagora, A., 802 (Y.F.)
——, with ——, 114 (Y.F.)
Lent, H., 973 (Tryp.)
—— & de Oliveira, S. J., 973 (Tryp.)
León, A. P., 193, 202, 984 (Typh.)
Leon y Blanco, F. & Angel Abalo, M., 225 (Der.)
—— & Oteiza, A., 585, 750 (Der.)
——, with Soberón y Parra, 1027 (Der.)
Leprosy in India, (291), 739, 1005 (Lep.)
Leprosy Review, (1013) (Lep.)
Leslie, A., with Silverman, 1001 (Am.)
Lester, H. M. O., 788 (Tryp.)

Leventhal, L., with Stryker & Halbeisen, 581 (Def. Dis.)
Lever, R. J. A. W., 778 (Mal.), 938 (Ent.)
Levine, H. D., 556 (Typh.)
Levkovich, E. N., 268 (Typh.)
—— & Petrishcheva, P. A., 268 (Typh.)
Levy, B. & Black-Schaffer, B., 833 (Der.)
Lewis, B. O., with Sodeman, 562 (Am.)
Lewis, D. J., 246 (Mal.), 1042 (Ent.)
Lewis, J. A., 405 (Ulc.)
Lewis, R. A., 443 (Mal.)
——, with Senekjie, 977 (Leish.)
Lewthwaite, R., 887, 979 (Typh.)
Lewy, R. B., 1035 (Oph.)
Li, C. C., with Pollitzer, 726 (Pl.)
Li, C. H., with Chang, Tong & Chin, 744 (Hel.)
Liebermann, H. R., 894 (Am.)
Lieu, V. T., 876 (Leish.)
Lillie, R. D., 414, 662 (Lab.)
Lima, S., with Soper, Wilson & Antunes, 79 (B.R.)
Lindgren, A., with Kaplan, 996 (Den.)
Lindquist, A. W., with Madden & Knipling, 73 (Ent.)
——, Schroeder, H. O. & Knipling, E. F., 1044 (Ent.)
Linhares, H., 277, 278 *bis*, 279, 373 (Y.F.), 309 (Vms.), 394, 395, 473, (904), 1014 (Lep.)
——, with Villela, (214) (Lep.)
Linskis, D. Y., 988 (Typh.)
Linton, R. W. & Jennings, R. K., 377 (Chl.)
——, with ——, 378 *bis*. (Chl.)
Lipman, B. L., Casey, A. V., Byron, R. A. & Evans, E. C., 371 (Typh.)
Lippincott, S. W., with Gordon, Marble, Ball, Ellerbrook & Glass, 692 (Mal.)
Lipscomb, L. L. & McMahon, J. L., 804 (Den.)
Lisbôa, A., 817 (Hel.)
Liu, W. T., 719 (Typh.)
Liu, Y. P., with Cheng & Tan, 733 (Am.)
Livesay, H. R. & Pollard, M., 203 (Typh.)
Lleras Restrepo, F., with Ignacio Chala, 566 (Lep.)
Loewenstein, A., with McGregor, 91 (Mal.)
Lofgren, R. C., 388 (Ys.)
Logue, J. B., 274 (Typh.)
Longley, E. O., 979 (Typh.)
Lopes, C. F. & Laender, J. F., 979 (Leish.)
Löscher, H., with Beiglböck, 31 (Typh.)
Loudon, J. R., with Jacoby, Wyne & Failmezger, 559 (Dys.)
Lourie, E. M., 559 (Am.)
Louw, A. & Nielsen, H. E., 60 (Haem.)
Low, G. C., 845 (Reports, etc.)
Lowe, J., 711 (Leish.)
Lozano Hube, E., with Mazzoti, 55 (Hel.)
Lucas, R. B., 886 (Typh.)
Lucena, D., with Magalhães, Coutinho, Gouvêa & Ignacio, 1016 (Hel.)
Luis Carri, E., (810) (Am.)
Luis Dao L., 17, 711 (Tryp.), 697 (Mal.)
Lurie, H. I., 824 (Haem.)
Lwoff, M. & Nicolle, P., (189) (Tryp.), (842) (Ent.)
——, with ——, (189) (Tryp.)
Lyon, E. & Kleinhaus, E. M., 1040 (Misc. Dis.)

M.

McAllister, J., with Augustine & Weinman, 288 (R.F.)
McCann, W. J., with Hirst, 838 (Misc. Dis.)
McCann, W. S., with Sellards, 115 (Y.F.)
McCarthy, D. D., with Annecke & Wilson, 966 (Mal.)
Macchiavello, A., 103 (Typh.)
—— & Uriguen, D., 466 (Pl.)
McClure, R. D. & Lam, C. R., 627 (Mal.)
McCombie, 405 (Ulc.)
McConaghey, R. M. S., 730 (Am.)
McCorkle, J. K., 536 (Mal.)
McCoy, O. R., (229) (Ent.), 433, 785 (Mal.)
McCreary, J. F. & Tisdall, F. F., 58 (Def. Dis.)
McCulloch, R. N., 369 (Typh.)
McDaniel, G. E., with Faust & Scott, 620 (Mal.)
McDermott, K. F., with LeFevre & Venner, 470 (Ys.)
Macdonald, G., 630, 965 (Mal.), 839 (Ent.)
McDonald, S., with Bingham, 305 (Hel.)
McDonald, S. F., 371 (Typh.)
McFarlane, A. L. & Branday, W. J., 755 (Misc. Dis.)
Macfie, J. W. S., 244 (Mal.)
McGann, V. G., with Burrows, Mather & Wagner, 727 (Chl.)
McGavack, T. H. & German, W. M., 141 (Haem.)
McGinn, S. & Carmody, J. T. B., 341 (Mal.)
Macgregor, I. M., 413 (Ent.)
McGregor, I. S. & Loewenstein, A., 91 (Mal.)
McGregor, M., 417 (Reports, etc.)
McGregor, T., with Irons, Bohls & Thurman, 462 (Typh.)
McHardy, G., with Browne & Spellberg, 729 (Am.)
MacHatton, R. M., with Chen & Geiling, 869 *bis*, 872 (Tryp.)
Machella, T. E. & Forrester, J. S., 994 (Typh.)
McIndoo, N. E., with Roark, 841 (Ent.)
Mackay-Dick, J., 377 (Pl.), (977) (Leish.)
McKee, R. W., with Ball, Anfinsen, Geiman & Ormsbee, 867 (Mal.)
Mackenzie, G. K., with Ferriman, 124 (Dys.)
McKibbin, J. M., with Hegsted & Stare, 784 (Mal.)
MacKichan, I. W., 13 (Tryp.)
Mackie, J. W., 417 (Reports, etc.)
Mackie, T. T., Hunter, G. W., III & Worth, C. B., 665 (B.R.)
McLaughlin, R. F., with Michael & Cenac, 66 (Der.)
McLaughlin, R. R. M., 1027 (Der.)
MacMahon, H. E. & DelVecho, S. B., 506 (Lab.)
McMahon, J. L., with Lipscomb, 804 (Den.)
McNaughton, F., with Anderson & Begg, 497 (Misc. Dis.)
Macpherson, A., with Findlay & Hill, 289 (Ys.)
Macquaide, D. H. G., 145 (Heat Str.)
McSweeny, A., with Flecker, 66 (Der.)
Madden, A. H., Lindquist, A. W. & Knipling, E. F., 73 (Ent.)

Maegraith, B. G., 10 (Bl), 64 (Vms.), 630 (Mal.), 788 (Bl.)
——, *et al.*, 780 (Mal.)
——, Brown, G. M., Rossiter, R. J., Irvine, K. N., Lees, J. C., Parsons, D. S., Partington, C. N., Rennie, J. L. & Havard, R. E., 251 (Mal.)
—— & Findlay, G. M., 12 (Bl.)
—— & Havard, R. E., 12 (Bl.), 863 (Mal.)
——, —— & Parsons, D. S., 968 (Bl.)
Magalhães, A., Coutinho, B., Gouvêa, L., Lucena, D. & Ignacio, L., 1016 (Hel.)
Magalhães, B. F. & Dias, C. B., 1016 (Hel.)
de Magalhães, O. & Rocha, A., 986, 988 (Typh.)
Magnuson, H. J., with Eagle, 356 (Tryp.)
Mahaffy, A. F., with Smithburn & Haddow, 321 (Misc. Dis.)
Mahmoud, A. H., 412 (Prot.)
Maievsky, with Ravikovich, Starovierova & Neuman, 549 (Typh.)
Maievsky, M. M., with ——, —— & Ratner, 549 (Typh.)
Mair, L. P., 944 (B.R.)
Majumder, D. N. & Das Gupta, C. R. (141) (Haem.)
Malhotra, R. P. & Chhuttani, P. N., 59 (Haem.)
Malone, R. H. & Brooks, A. G., 357 (Leish.)
Mandekos, A., 501 (Ent.)
Mangabeira, O., Jr. & Galindo, P. (151) (Ent.)
——, with Pondé & Jansen, (876) (Leish.)
Mann, I., 826 (Haem.)
Manson-Bahr, P., 10 (Bl.), 176 (Mal.), 207, 559 (Am.), 762 (Misc.), 845 (Reports, etc.)
—— & Muggleton, W. J., 325 (Ent.), 434 (Mal.)
Manson-Bahr, P. E. C. & Guyer, O. K. G., 409 (Misc. Dis.)
Mantilla, L. R., with Rivas & Góbich, 910 *bis* (Hel.)
Manwell, R. D., Coulston, F., Binckley, E. C. & Jones, V. P., 839 (Prot.)
Mao, C. P., with Yu, 747 (Hel.)
Marble, A., with Gordon, Lippincott, Ball, Ellerbrook & Glass, 692 (Mal.)
Marchionini, A., 310 (Der.)
Mariani-Tosatti, G., 498 (Misc. Dis.)
Mariano, J., 133, 1010 (Lep.)
——, with de Oliveira Castro, 1009 (Lep.)
——, with de Souza-Araujo & de Oliveira Castro, 1009 (Lep.)
Mariotte, C. O., Bustamante, M. E. & Varela, G., 721 (Typh.)
Markell, E. K., 468 (Am.)
Markson, J. L., with Findlay & Holden, 6, 350 (Mal.)
Marneffe, H., Ranque, J. & Sautet, J., 247 (Mal.)
——, with Sautet, 336 (Mal.)
Marques, A., 209 (R.F.)
Marriott, H. L., 753 (Misc. Dis.)
Marsh, F., 493 (Heat Str.)
—— & Wilson, H. A., 588 (Ulc.)
Marshall, J. M., 438 (Mal.)
——, with Walters, Watkins & Butt, 37 (Am.)
Martin, A. P., 78 (B.R.)
Martin, H., with Länger & Muller, 499 (Ent.)
—— & Wain, R. L., 152 (Ent.)
Martin, L. A., with Blanc & Baltazard, 713 (Typh.)
Martin, R., (324), 414 (Ent.)
—— & Bebey, M., 37 (Am.)
Martin, W. B., with Coggeshall & Bates, 698 (Mal.)
Martins, A., Versiani, V. & Tupínambá, A., (875) (Tryp.)
Marx, R., with Aschenbrenner, 105 (Typh.)
Masen, J. M., 8 (Mal.)
Mason, A. S., with Ward, 934 (Ulc.)
Mason, E. D., 227 (Heat Str.)
Master, A. M., 657 (Misc. Dis.)
Mater, D. A., with Freis, 402 (Sp.)
Mather, A. N., with Burrows & Wagner, 728 (Chl.)
——, with ——, —— & McGann, 727 (Chl.)
Mathieson, D. R., with Stoll & Ward, 999 (Am.)
——, with Wilson & Jachowski, 73 (Ent.)
Matoff, K., 579 (Hel.)
Mattingly, P. F., 434 (Mal.)
Maveety, H. M., Turnbull, R. B., Jr., & Bauer, C. R., 441 (Mal.)
Maxcy, K. F., with Blake, Sadusk, Kohls & Bell, 991, 993 (Typh.)
May, W. K. C., 735 (R.F.)
Mayer, M. M., with Heidelberger, 689 (Mal.)
Mayer, R. A., with Di Giacomo, 47 (Hel.)
Mazza, S., Miyara, S. & Jörg, M. E., 792 (Tryp.)
Mazzotti, L., 134, 219, 398, 647, (926) (Hel.)
—— & Lozano Hube, E., 55 (Hel.)
Mead, J. & Koepfli, J. B., 9 (Mal.)
Measham, J. E., Yoeli, M. & Finnegan, J. D., 695 (Mal.)
Medical Clinics of North America, 765 (B.R.)
Medical Research Council, 843 (Lab.), 1052 (B.R.)
Medlicott, R. W., 581 (Def. Dis.)
Megaw, J. W. D., 506 (Reports, etc.), 797 (Typh.)
de Meillon, B., Golberg, L. & Lavoipierre, M., (1043) (Ent.)
——, with —— & ——, (1043) (Ent.)
——, Parent, M. & Black, L. O'C., (940) (Ent.)
Meira, J. A. & Ramos, J., Jr., 572 (Hel.)
de Meira, M. T. V. & Ferreira, T. G., (1044) (Ent.)
Mellanby, K., 839 (Ent.)
de Mello, R. P., with Cruz, 1020 (Hel.)
Mendelsohn, H. V., 66 (Der.)
Menon, I. G. K., 589 (Misc. Dis.)
Menon, M. C. & Ibbotson, C., 884 (Typh.)
Menzies, T. H., with Anderson, von Deschwanden & Gray, 961 (Mal.)
Merino, C., with Hurtado & Delgado, 824 (Haem.)
Merrill, B. R., 691 (Mal.)
Merrill, E. D., 234 (B.R.)
Mestre Miyares, J. J., 43 (Lep.)
Metcalf, R. J. & Ungar, J., Jr., 341 (Mal.)
Metcalf, R. L. & Hess, A. D., 351 (Mal.)
——, with Kruse & Hess, 184 (Mal.)
Meyer, H., with Romaña, 972 (Tryp.)
Meyer, K., with von Sallmann & Di Grandi, 489 (Oph.)
Michael, P., McLaughlin, R. F. & Cenac, P. L., 66 (Der.)

Mickelsen, O., with Henschel, Taylor, Brozek & Keys, 145 (Heat Str.)
Miles, V. I., with Coatney & Cooper, 865 (Mal.)
Miller, H., (411) (Misc. Dis.)
Miller, J. F. & Einhorn, N. H., 578 (Hel.)
——, with —— & Whittier, 911, 912, 914 (Hel.)
Miller, M. H., with Whitehill, 576 (Hel.)
Mills, C. A., 930 (Heat Str.)
Milne, R. I., (996) (Typh.)
Mina, H., 494 (Ulc.)
Mingoja, Q., 632 (Mal.)
Minnich, V., with Moore, Vilter & Spies, 482 (Haem.)
Minton, J., 1037 (Oph.)
Miranda, R. N., 1011 (Lep.)
——, with de Souza-Araujo, 1007 (Lep.)
Misra, K. N., 120 (Chl.)
Miterstein, B. & Stern, H. J., 1030 (Oph.)
Mitscherlich, E., 78 (Reports, etc.)
Miyara, S., with Mazza & Jörg, 792 (Tryp.)
Mohammed, A. H., 583 (Vms.)
Moiser, B., 294 *bis* (Lep.)
Mole, R. H., 1023 (Haem.)
Mom, A. M. & Basombrio, G., 1012 (Lep.)
Monchadskiĭ, A. S., with Blagoveshchenskiĭ & Bregetova, 230 (Ent.)
Money, T. D. F., 392 (Lep.)
Mönnig, H. O., 401 (Hel.)
Monro, H. A. U., Briand, L. J., Delisle, R. & Smith, C. C., 759 (Ent.)
Monthly Bull. Ministry of Health & Emergency Pub. Health Lab. Service, 680 (Rab.), 879 (Typh.)
Montoya, J. A., 551 (Typh.)
Montpellier, J. & Catanei, A., 67 (Der.)
Moore, C. V., Vilter, R., Minnich, V. & Spies, T. D., 482 (Haem.)
Mooser, H., 500 (Ent.)
Moragues, V., with Greiff & Pinkerton, 362 (Typh.)
Morales Villazon, N., with Savino & Kuhn, 558 (Pl.)
Moran, T. J., with Fetterman & Hess, 495 (Misc. Dis.)
Morehead, J. F., 720 (Typh.)
Morgan, C. N., 124 (Am.)
Morris, J. N., 941 (Reports, etc.)
Morris, T. L., with Hynes & Ishaq, 651 (Haem.)
Morrison, D., with Greenslade, (229) (Prot.)
Morrison, D. B., with Dulaney, 352 (Mal.)
Morton, H. E. & Engley, F. B., Jr., 997 (Dys.)
Morton, T. C. St. C., 731 (Am.)
Moshkovski, S. D., (622) (Mal.)
Mouritz, A., (814) (Lep.)
Moustardier, G. & Nicol, R., 933 (Ulc.)
Mozley, A., 516 (B.R.)
Mudrow, L. & Schultz, F., 814 (Lep.)
Muggleton, W. J., with Manson-Bahr, 325 (Ent.), 434 (Mal.)
Mugrage, E. R., with Florio & Stewart, 118 (Den.)
——, with Stewart & Florio, (119) (Den.)
Muir, E., 390 *ter*, 1004 (Lep.)
Mukerji, A. K. & Bhaduri, N. V., 919 (Hel.)
Mukerji, B., with Pal, Gupta & Chatterjee, 286 (Am.)
Mukherji, N., with Dharmendra, 900, 903 (Lep.)
Mülhens, K., 502 (Ent.)
Müller, H. K., 548 (Typh.)
Muller, P., with Länger & Martin, 499 (Ent.)
Mulrennan, J. A., Goodwin, M. & Shannon, R. C., 633 (Mal.)
Muniz, J., & De Freitas, G., 975 (Tryp.)
Munter, E. J., 805 (Pl.)
Murphy, R. C., Jr. & Shapiro, S., 58 (Haem.)
Murray, E. S., with Yeomans, Snyder, Zarafonetis & Ecke, 201 (Typh.)
Mushett, C. W., with Siegel, 178 (Mal.)
——, with —— & Emerson, 628 (Mal.)

N.

Nabokov, V. A., 1045 (Ent.)
Naftalin, J. M., with Graham, 876 (Leish.)
Nagibina, N. I., with Yarygin, 349 (Mal.), 989 (Typh.)
Naidu, V. R., with Heilig, 30 (Typh.)
Naiman, D. N., (100) (Tryp.)
Nájera Angulo, L., 130 (R.F.)
Najjar, V. A., with Roberts, 56 (Def. Dis.)
Napier, L. N., 79 (B.R.)
Nargund, K. S., with Paranjape, Phalnikar & Bhide, 571 (Hel.)
Nasi, K. W., with Hundley, 282 (Pl.)
Natarajan, B., with Viswanathan, 411 (Misc. Dis.)
Neel, J. V., with Valentine, 223, 825 (Haem.)
Neeman, M., (91) (Mal.)
Neghme, A., 453 (Tryp.)
Negroni, P. & Bonfiglioli, H., 586 (Der.)
Nelson, J. H. & Cruickshank, J. C., 363 (Typh.)
Nelson, N., with Eichna, Bean & Ashe, 751 (Heat Str.)
Nery Guimarães, F. & Jansen, G., 972 (Tryp.)
Ness, A. T., with Brady, Lawton, Cowie, Andrews & Ogden, 747 (Hel.)
Nettel, R., 54 (Hel.)
Neuda, P. M. & Rosen, M. S., 825 (Haem.)
Neuman, A. S., with Ravikovich, Starovierova & Maievsky, 549 (Typh.)
——, with ——, ——, —— & Ratner, 549 (Typh.)
Nicol, L., with Ramon, Boquet, Richou & Delaunay-Ramon, (584) (Vms.)
Nicol, R., with Moustardier, 933 (Ulc.)
Nicolle, P. & Lwoff, M., (189) (Tryp.)
——, with ——, (189) (Tryp.), (842) (Ent.)
Nielsen, H. E., with Louw, 60 (Haem.)
Niesche, F. W., with Hughes, (628) (Mal.)
Nigeria, 790 (Tryp.)
Niño, F. L., 473, 909 (Hel.)
Nørgaard, F., with Petri & Kiaer, (308) (Def. Dis.)
Norris, R. F., with Kern, 341 (Mal.)
North, J. P., with Ravdin, (964) (Mal.)
Notestein, F. W., 507 (Reports, etc.)
Nutrition Reviews, New York, 8 (Mal.)
Nyasaland Protectorate, 588 (Ulc.)

O.

Obermayer, M. E. & Frost, K., 832 (Der.)
O'Brien, J. P., with Allen, 313 (Heat Str.)
O'Bryan, B. E., with Schuhardt, 387, 735, (R.F.)
O'Connor, N., 218 (Hel.)
Offenhauser, W., with Kahn & Celestin, 661 (Ent.)
Officer, R., 688 (Mal.)
Ogden, G. E., with Brady, Lawton, Cowie, Andrews & Ness, 747 (Hel.)
Olarte, J., with Varela & Castro Estrada, 927 (Der.)
Olavarria, J., with Clavero, (859) (Mal.)
Oldfield, M. C., (250) (Mal.)
Oleinik, E., with Kligler, 23 (Typh.)
de Oliveira, J. C. & de Bettencourt, A., 166 (Rab.)
de Oliveira, S. J., with Lent, 973 (Tryp.)
de Oliveira Castro, G. M., 960 (Mal.)
——, & Mariano, J., 1009 (Lep.)
——, with de Souza-Araujo & Mariano, 1009 (Lep.)
Oliver-González, J., 353 (Bl.)
—— & Bercovitz, Z. T., 219 (Hel.)
—— & Torregrosa, M. V., 45 (Hel.)
Olsen, O. W., (302) (Hel.)
Olson, T. A. & Keegan, H. L., (504) (Ent.)
Ordman, D., 41 (R.F.)
Ormsbee, R.A., with Ball, Anfinsen, Geiman & McKee, 867 (Mal.)
Osburn, H. S., 285 (Am.)
Osburn, L. W., 455 (Typh.)
Osimani, J. J., with Isola, (230) (Ent.)
Osorno-Mesa, E., with Boshell-Manrique, 114 (Y.F.)
Ostrovskaya, S. G., with Vainberg, Gulamova, & Perekriostova, 549 (Typh.)
Oteiza, A., 584 (Der.)
——, with Léon-Blanco, 585, 750 (Der.)
Ott, W. H., with Seeler, 540 (Mal.)
Ouary, G., with Hamm, 495 (Ulc.)
Ovchinnikova, L. M., (595 *bis*) (Ent.)
Ozburn, R. H., (229) (Ent.)

P.

Packchanian, A., (74) (Ent.)
Page, R. C., with Kleinman & Preisler, (829) (Vms.)
Pai, M. N., 942 (Reports, etc.)
Pakenham-Walsh, R., 400 (Hel.)
Pal, J. C., Mukerji, B., Gupta, J. C. & Chatterjee, M. L., 286 (Am.)
Palmer, E. D., 135 (Hel.)
Pampana, E. J., (533) (Mal.)
Pang, H. Q., with Lee, 593 (Misc. Dis.)
Panja, G., 892 (Chl.)
Panthier, R., with Giroud, 104, 985 (Typh.)
Pappenheimer, A. M., Thompson, W. P., Parker, D. D. & Smith, K. E., 826 (Haem.)
Paraense, W. L., (968) (Mal.)
Paramore, R. H., 452 (Bl.)
Paranjape, K. D., Phalnikar, N. L., Bhide, B. V. & Nargund, K. S., 571 (Hel.)
Parent, M. & Demoulin, M. L. (688) (Mal.)
——, with ——, 845 (Lab.)
Parent, M., with de Meillon & Black, (940) (Ent.)
Parish, H. J., 979 (Typh.)
Park-Ross, M., with Alexander & Stein, 38 (Am.)
Parker. D. D., with Pappenheimer, Thompson & Smith, 826 (Haem.)
Parker, R. R., with Steinhaus, 375 (Den.)
Parmakson, P., 295 (Lep.)
Parnet, J., with Sohier & Chon, (795) (Typh.)
Parrot, L., Catanei, A. & Collignon, E., 445 (Mal.)
—— & de Jolinière, P. B., (662) (Ent.)
Parry, E., (286) (Am.)
Parsons, B. T., with Theodor, 1043 (Ent.)
Parsons, D. S., with Maegraith, Brown, Rossiter, Irvine, Lees, Partington, Rennie & Havard, 251 (Mal.)
——, with —— & Havard, 968 (Bl.)
Parsons, R. J. & Zarafonetis, C. J. D., 654 (Der.)
Partington, C. N., with Maegraith, Brown, Rossiter, Irvine, Lees, Parsons, Rennie & Havard, 251 (Mal.)
Passalacqua, L. A., with Smith, 696 (Mal.)
Passmore, R., with Hardy, (977) (Leish.)
Patel, S. V., 448 (Mal.)
Patel, J. C., 263 (Leish.)
Patel, N. D., 754 (Misc. Dis.)
Paterson, J. C. S., with Ransome & Gupta, 146 (Misc. Dis.)
Patiño-Camargo, L., 265 (Typh.)
——, de Zulueta, J. & Toro, G., 799 (Typh.)
Patnam, P., with Russell, Knipe & Ramachandra Rao, 687 (Mal.)
Pattanayak, G. C., 405 (Ulc.)
Patterson, H. M., 42 (R.F.)
Patterson, H. S., 28 (Typh.)
Paul, B. M. & Chatterjee, B. C., 379 (Chl.)
Paul, J. R., with Philip & Sabin, 119 (Den.)
Paul, W. D., Antes, E. H. & Sahs, A. L., 804 (Den.)
Paulley, J. W. & Aitken, G. J., 55 (Def. Dis.)
Pavlovskii, E. N., 564 (R.F.)
—— & Aluimov, A. Y., 565 (R.F.)
Pavlovsky, E. N., 564 (R.F.)
Payne, A. M. M., 381, 1001 (Am.)
Peel, E., with van Hoof & Henrard, 96 (Tryp.)
Peixoto, P. G., 296 (Lep.)
Peña, L. S., with Alvarado, (448) (Mal.)
Pepper, D. S., with Fitz-Hugh & Hopkins, 340 (Mal.)
Perekriostova, E. P., with Vainberg, Gulamova & Ostrovskaya, 549 (Typh.)
Pereperez y Palau, F., 171 (Mal.)
Pérez Gallardo, F., with Clavero, 794 (Typh.)
——, with Gallardo & Sanz, 23 (Typh.)
Perkins, R. M., with Pope, 403 (Vms.)
Perlowagora, A. & Lennette, E. H., 114 (Y.F.)
——, with ——, 802 (Y.F.)
Perlzweig, W. A., with Cayer & Ruffin, 580 (Def. Dis.), 1021 (Sp.)
Peryassú, A., 688 (Mal.)
Pessôa, S. B. & Villela, F., 712 (Leish.)
Pestana, B. R., 638 (Typh.)
Peters, J. P., 227 (Heat Str.)
Petersen, M. C. & Fahey, J., 920 (Hel.)
Petri, S., Nørgaard, F. & Kiaer, W., (308) (Def. Dis.)

Petrishcheva, P. A., with Levkovich, 268 (Typh.)
Phalnikar, N. L., with Paranjape, Bhide & Nargund, 571 (Hel.)
Philip, C. B., with Kohls, Armbrust & Irons, 993 (Typh.)
——, Paul, J. R. & Sabin, A.B., 119 (Den.)
di Pietro, A., (1028) (Der.)
Pifano, C. F., 191 (Leish.)
Pigoury, L., 400 (Hel.)
——, with Poursines, (189) (Tryp.)
——, with ——, Borde & Bernard, (189) (Tryp.)
Pilcher, J. F., with Stewart, 712 (Leish.)
Piña Daza, M., 47 (Hel.)
Pinkerton, H., 878 (Typh.)
——, with Greiff & Moragues, 362 (Typh.)
Pinto, C., (875) (Tryp.)
Pirk, L. A. & Engelberg, R., 964 (Mal.)
Pirot, R. & Bourgain, M., 137 (Hel.), 811 (R.F.)
Pita Gandarias, D. E., 170 (Mal.)
Pitão, J. S., (749) (Def. Dis.)
Pittaluga, G., 803 (Den.)
Pitts, G. C., Johnson, R. E. & Consolazio, F. C., with Poulin, J., Razoyk, A. & Stachelek, J., 226 (Heat Str.)
Placeo, F., 38 (Am.)
Platt, R., 588 (Heat Str.)
Plotz, H., Bennett, B., Wertman, K. & Snyder, M., 636 (Typh.)
——, with Hamilton & Smadel, 796 (Typh.)
Plyasena, A. K., with Samarasinghe & Rustomjee, (245) (Mal.)
Podyapolskaya, V. P. & Isaicheva, A. I., 573 (Hel.)
Pogge, R. C., with Faget, 297, 646 (Lep.)
Poirier, M., (214) (Lep.)
Polakow, R., 41 (R.F.)
Pollack, G. R., with van Rooyen, Darskin & Bearcroft, 194 (Typh.)
Pollard, M. & Augustson, G. F., 551 (Typh.)
——, with Livesay, 203 (Typh.)
Pollitzer, R. & Li, C. C., 726 (Pl.)
——, with Shih, 725 (Pl.)
Pondé, R., Mangabeira, O., Jr. & Jansen, G., (876) (Leish.)
Pons, R., 335 (Mal.)
Pope, C. H. & Perkins, R. M., 403 (Vms.)
Porter, R. J., with Coggeshall & Laird, 699 (Mal.)
Poth, E. J. & Ross, C. A., 35 (Dys.)
Potter, H. W., Bronstein, L. H. & Gruber, C. M., 690 (Mal.)
Potts, W. H., 98 (Tryp.)
—— & Vanderplank, F. L., 869 (Tryp.)
Poulin, J., with Pitts, Johnson, Consolazio, Razoyk & Stachelek, 226 (Heat Str.)
Poursines, Y. & Pigoury, L., (189) (Tryp.)
——, ——, Borde, R. & Bernard, M., (189) (Tryp.)
Pozzo, A. A., (34) (Pl.)
Pradhan, K. G., 29 (Typh.)
Preisler, P. W., with Kleinman & Page, (829) (Vms.)
Prendergrass, R. C., with Kunstadter, (655) (Der.)
Pretoria Leper Institution, 292 (Lep.)
Price, E. W., 1037 (Ulc.)
Priest, P., 559 (Am.)
Proc. Ann. General Meeting of Assam & Northern Bengal Branch Brit. Med. Ass., Shillong, 405 (Ulc.)
Proc. Roy. Soc. Med., 979 (Typh.)
Prout, C., with Hindle, Rose & Trevett, 446 (Mal.)
Pshenichnov, A. V., 269 (Typh.)
—— & Khramushin, J. E., 324 *bis* (Ent.)
Public Health Rep., 32, 33 (Pl.), 757 (Ent.)
Pulvertaft, R. J. V., with Adler, 412 (Prot.)
Puri, I. M., 85 (Mal.)
Purohit, G. R., 64 (Vms.)

Q.

Quill, L. M. & Burch, J. C., 225 (Der.)
Quinn, G. P., with Hiatt, 778 (Mal.)
Quiros, M., with Kessel, Allison, Kaime & Gloeckner, 40 (Am.)
Qutubuddin, M., with Farooq, 978 (Leish.)

R.

Rachmilewitz, M. & Braun, K., 821 (Def. Dis.)
Radice, J. C., with Segura, Donin & Giusti, (141) (Haem.)
Raettig, H., 25 (Typh.)
Raevsky, G. E., 858 (Mal.)
Rai Chaudhuri, M. N., with Chaudhuri, 221 (Sp.), (838) (Misc. Dis.), 962 (Mal.)
Raïkher, B. I., 270 (Typh.)
Raina, B. L., (967) (Mal.)
Rajagopalan, S., with Dharmendra, 1012 (Lep.)
Rake, G., with Jones & Hamre, 543 (Leish.)
Rakshit, P., with Bose, 451 (Mal.)
——, with Bose & Ghosh, 444 (Mal.), 468 (Am.)
Ramachandra Rao, T., with Russell, Knipe & Patnam, 687 (Mal.)
Raman, T. K., 262 (Leish.)
Ramon, G., Boquet, P., Richou, R., Nicol, L. & Mme. Delaunay-Ramon, (584) (Vms.)
Ramos, A. S., with Corrêa, (173), 961 (Mal.)
Ramos, J., Jr., with Meira, 572 (Hel.)
Ramsey, E. M., with Temkin, 533 (Mal.)
Randall, T., 755 (Misc. Dis.)
Randerath, E., 458 (Typh.)
Ranque, J., with Marneffe & Sautet, 247 (Mal.)
Ransome, G. A., Gupta, L. M. & Paterson, J. C. S., 146 (Misc. Dis.)
Ranta, L. E. & Dolman, C. E., 467 (Chl.)
Rao, G. R. (620) (Mal.)
Rao, M. V. R., Colah, R. B. M. & Kalle, R. A., 932 (Ulc.)
Rao, T. R., with Viswanathan & Rao, 625 (Mal.)
Rao, T. S. R., with Viswanathan & Rao, 625 (Mal.)
Ratner, S. M., with Ravikovich, E. M., Starovierova, Neuman & Maievsky, 549 (Typh.)
Raúl Larenas, M., with Beltrán, 20 (Leish.), 36 (Am.), 594 (Prot.)
——, with Guerra, Beltrán & De la Garza, 866 (Mal.)
Ravdin, I. S. & North, J. P., (964) (Mal.)

Raven, R. W., 106 (Typh.)
Ravikovich, E. M., Starovierova, A. G., Neuman, A. S. & Maievsky, 549 (Typh.)
——, ——, ——, —— & Ratner, S. M., 549 (Typh.)
Raymond, W. D., 250 (Mal.), 498 (Misc. Dis.)
Razoyk, A., with Pitts, Johnson, Consolazio, Poulin & Stachelek, 226 (Heat Str.)
Reardon, L. V., with Rees, Bozicevich & Daft, 36 (Am.)
Rebelo, A., with Foy, Kondi & Soeiro, 634 (Bl.)
Rector, N. H., 185 (Mal.)
Redmond, W. B., 95 (Mal.)
Reed, H., 1032 (Oph.)
Rees, C. W., Bozicevich, J., Reardon, L. V. & Daft, F. S., 36 (Am.)
Reeves, W. C., 437 (Mal.)
Reid, J., 630 (Mal.)
——, with Drew, 862 (Mal.)
Reller, H., with Tsuchiya, 817 (Hel.)
Remington, R. E., 307 (Def. Dis.)
Remlinger, P., 679 (Rab.)
—— & Bailly, J., 169, 170 (Rab.)
Renault, L., with Versiani, (209) (Am.)
Renkonen, K. O., 621 (Mal.)
Rennie, J. K., with Beach, 880 (Typh.)
Rennie, J. L., with Brown, 780 (Mal.)
——, with Maegraith, Brown, Rossiter, Irvine, Lees, Parsons, Partington & Havard, 251 (Mal.)
Renoux, G., with Durand, Beguet & Horrenberger, 107 (Typh.)
——, with Horrenberger, 107 (Typh.)
Reuter, A., 204 (Typh.)
—— & Schäfer, W., 204 (Typh.)
Rewell, R. E., (1022) (Sp.)
Ribbands, C. R., 3 *bis*, 245 (Mal.)
Rice, F. A. H., with Young, 415 (Lab.)
Rice, M. E., with Watson, 173 (Mal.)
Rice, P. L., Huffaker, C. B. & Back, R. C., 632 (Mal.)
Richards, C. S., with Scott & Seaman, 917 (Hel.)
Richardson, F. MacD., with Diasio, 205 (Den.)
Richou, R., with Ramon, Boquet, Nicol & Delaunay-Ramon, (584) (Vms.)
Richter, C. P., 811 (R.F.)
Rico, B. C., 177 (Mal.)
Rico-Avello, C., (6) (Mal.)
Ridley, H., 583 (Vms.)
Riemerschmid, G., 317 (Heat Str.)
Rifkin, H., 645 (Ys.)
Rigdon, R. H., 250, 540, 689 (Mal.)
Ritchie, E. A. A., (149) (Misc. Dis.)
Ritter, C., 134 (Hel.)
Rivas, C. I., Góbich, E. & Mantilla, L. R., 910 *bis* (Hel.)
de Rivas, D., 38 (Am.)
Rivenburg, H., with Barlow & Auerbach, 443 (Mal.)
Rivera Lugo, I., (354) (Bl.)
Roark, R. C., 841 *bis* (Ent.)
—— & McIndoo, N. E., 841 (Ent.)
Robb, J. V. M., with Browning & Browning, 258 (Tryp.)
Robbers, H., (106) (Typh.)
Roberts, D. W. & Najjar, V. A., 56 (Def. Dis.)
Roberts, J. I., with Wright, 215 (Hel.)
Robertson, J. D., 860 (Mal.)
Robertson, J. N., 439 (Mal.)
Robinson, G. G. (151) (Ent.)
Rocha, A., with de Magalhaes, 986, 988 (Typh.)
Rodhain, J., 820, 821 (Hel.), (834) (Der.)
—— & Barlovatz, A., 887 (Typh.)
—— & van Goidsenhoven, C., 869 (Tryp.)
—— & van Hoof, M. T., (778) (Mal.), (791) (Tryp.)
—— & van den Berghe, L., 872 (Tryp.)
Rodriguez de la Cruz, J. M., with Roig, 214 (Lep.)
Rodriguez-Molina, R., with Bercovitz, Hargrave, Dickie & Green, 47 (Hel.)
——, with —— & Shwachman, 572 (Hel.)
Rogers, L., 282 (Chl.), 630 (Mal.)
Rogers, W. P., 299, (577) (Hel.)
Roig, J. T. & Rodriguez de la Cruz, J. M., 214 (Lep.)
Romaña, C., 873, 974 (Tryp.)
—— & Cossio, F., 874 (Tryp.)
—— & Dias, E., 974 (Tryp.)
—— & Meyer, H., 972 (Tryp.)
Romero Moliner, R., 180 (Mal.)
van Rooyen, C. E., Bowie, J. H. & Krikorian, K. S., 264 (Typh.)
—— & Danskin, D., 463 (Typh.)
——, with Darskin, D., Pollack, G. R. & Bearcroft, W. G. C., 194 (Typh.)
Rose, A. S., with Hindle, Trevett & Prout, 446 (Mal.)
Rose, G., 537 (Mal.)
Rose, H. M., 454 (Leish.)
——, with Culbertson, 136 (Hel.)
Rose, T. F., 931 (Heat Str.), 997 (Dys.)
Rosen, M. S., with Neuda, 825 (Haem.)
Rosenberg, A. A., 440 (Mal.)
Ross, C. A., with Poth, 35 (Dys.)
Ross, H., 297 (Lep.)
Ross, J. A., 38 (Am.)
——, with Eccles, 297 (Lep.)
Rossi, M., with Battelli & Coceani, 977 (Leish.)
Rossiter, R. J., with Maegraith, Brown, Irvine, Lees, Parsons, Partington, Rennie & Havard, 251 (Mal.)
Rotberg, A., 568 (Lep.)
——, with Bechelli & Keil, 900 (Lep.)
Roth, V. E., 719 (Typh.)
Roy, A. C., (829) (Vms.)
Roy. Nav. Med. Bull, 179 (Mal.)
Royston, G. R., with Lamb, 561 (Am.)
Rozeboom, L. E. & Hess, A. D., 182 (Mal.)
Ruffin, J. M., with Cayer & Perlzweig, 580 (Def. Dis.), 1021 (Sp.)
Ruge, H. & Kremer, H., 85 (Mal.)
Ruiloba, J., with González Ochoa, 142 (Der.)
Ruiz Moreno, F., (366) (Typh.)
Ruiz Sánchez, F., 272 (Typh.)
Ruska, H., with Eyer, 23 (Typh.)
Russ, S. E. & Gaynor, J. S. (533) (Mal.)
Russell, H. K., 630 (Mal.)
Russell, P. F., Knipe, F. W., Ramachandra Rao, T. & Patnam, P., 687 (Mal.)
Rustomjee, K. J., (620), 684 (Mal.)
——, with Samarasinghe & Plyasena, (245) (Mal.)
Ryan, R. C., with Ball, 19 (Leish.)
Ryjov, N. V., 459 (Typh.)

S.

Sabin, A. B., with Philip & Paul, 119 (Den.)
—— & Schlesinger, R. W., 891 (Den.)
Sabine, J. C., with Wright, 252 (Mal.)
Sabrosky, C. W. & Usinger, R. L., 244 (Mal.)
Sadusk, J. F., Jr., with Blake, Maxcy, Kohls & Bell, 991, 993 (Typh.)
Sagher, F., 740 (Lep.)
Sahs, A. L., with Paul & Antes, 804 (Den.)
St. Amant, L. S., with Byrd & Bromberg, 479 (Hel.)
Sal y Rosas, F., (327) (Reports, etc.)
Saliternik, Z., with Shapiro & Belferman, 247 (Mal.)
von Sallmann, L. & Meyer, K., with Di Grandi, J., 489 (Oph.)
Salm, G., with Gaud & Fassi, 420 (Reports, etc.)
Samaniego, B. M., 889 (Bart.)
Samarasinghe, D. S., Plyasena, A. K. & Rustomjee, K. J., (245) (Mal.)
Sampayo, R. R. L., 65 (Vms.)
Sams, C. F., 147 (Misc. Dis.)
Samuels, R. D., with Abraham, 619 (Mal.)
Sanabria, A., 396 (Hel.)
Sandy, F., with Whitelaw, (304) (Hel.)
Sandza, J. G., with Balamuth, 999 (Am.)
Santra, I., 393 (Lep.)
——, with Dharmendra, 738 (Lep.)
Sanz, J., with Gallardo & Perez Gallardo, 23 (Typh.)
Sapeika, N., 153 (Reports, etc.)
Sarmento, A., 1015 (Hel.), (1025) (Haem.)
Sarrouy, J., Dendale, R., Combe, P., Arnaud, R. & Gillot, F., 102 (Leish.)
—— & Gillot, F., 101 (Leish.)
Sartorius, K., (915) (Hel.)
Sather, R. O. & Silberstein, J. S., 885 (Typh.)
Saunders, G. F. T., Holden, J. R. & Hughes, M. H., 354 (Tryp.)
Sautet, J. & Marneffe, H., 336 (Mal.)
——, with —— & Ranque, 247 (Mal.)
Savino, E. & Goobar, J. K., 33, 466 (Pl.)
——, Kuhn, M. J. & Morales Villazon, N., 558 (Pl.)
Sayer, A., (67) (Der.)
Schäfer, W., with Reuter, 204 (Typh.)
Schäffer, K., 857 (Mal.)
Schechter, M. S., with Hall & Fleck, 842 (Ent.)
Schell, S. C., with Schoof & Ashton, 626 (Mal.)
Scheller, H., 200 (Typh.)
Scherer, J. H. & Cecil, R. C., 583 (Haem.)
Schlesinger, R. W., with Sabin, 891 (Den.)
Schlumberger, H. G., 655 (Der.)
Schnakenbeck, W., 321 (Misc. Dis.)
Schnelle, G. B. & Young, R. M., 52 (Hel.)
Schoenaers, F., with van Goidsenhoven, 791 (Tryp.)
Schoof, H. F., 537 (Mal.)
——, Schell, S. C. & Ashton, D. F., 626 (Mal.)
Schramm, M., 411 (Misc. Dis.)
Schroeder, H. O., with Lindquist & Knipling, 1044 (Ent.)
Schröer, W., 545 (Typh.)
Schubert, R., (304) (Hel.)
Schüffner, W. & Swellengrebel, N. H., 922, 924 (Hel.)
Schuhardt, V. T., & O'Bryan, B. E., 387, 735 (R.F.)
Schulten, H., 27 (Typh.)
Schultz, F., with Mudrow, 814 (Lep.)
Schwartz, I., with Kligler & Guggenheim, 930 (Heat Str.)
Schwers, G. A., 846 (Reports, etc.)
Schwetz, J., with Baumann, H. & Fort, M., 810 (R.F.)
—— & Dartevelle, E., (815 *bis*), (1017) (Hel.)
Scodeller, J. A., with Videla (816) (Hel.)
Scott, H. H., 559 (Am.), 839 (Ent.), 845 (Reports, etc.)
Scott, J. A., with Faust & McDaniel, 620 (Mal.)
Scott, O. K., Richards, C. S. & Seaman, E. A., 917 (Hel.)
Scott, R. B., 42 (R.F.), 728 (Dys.)
Scovel, F. G., 875 (Leish.)
Seager, L. D., 261 (Tryp.)
Seaman, E. A., with Scott & Richards, 917 (Hel.)
Seeler, A. O., 699 (Mal.)
—— & Ott, W. H., 540 (Mal.)
Segal, M., 897 (Am.)
Segura, G., Radice, J. C., Donin, L. & Giusti, C. L., (141) (Haem.)
Sellards, A. W. & McCann, W. S., 115 (Y.F.)
Selman, D., 816 (Hel.)
Sen, A. N., & Basu, U. P., 893 (Chl.)
Sen, J. B., 1014 (Lep.)
Sen, K., with Kirwan & Bose, 1036 (Oph.)
Sen, S., with Basu, 645 (R.F.)
Sen Gupta, P. C., (264), 358, 542, 543 (Leish.), 557 (Typh.)
Sen Gupta, S. B., with Das Gupta & Banerjee, 129 (Am.)
Senekjie, H. A., 130 (R.F.), 359 (Leish.)
—— & Lewis, R. A., 977 (Leish.)
Sergent, Ed., (75), (326) (Reports, etc.)
—— & Béguet, M., 270 (Typh.)
—— & Horrenberger, R., 365 (Typh.)
Sergent, Et., 141, 404 *ter.*, 405 (Vms.)
Sergiev, P. G. & Tiburskaya, N. A., 622 (Mal.)
Serial, A., with Fernández, Vaccaro & Carboni, 832 (Der.)
Severens, J. M. & Tanner, F. W., 847 (Reports, etc.)
Sevringhaus, E. L., 308 (Def. Dis.)
Shama Rao, A., 393 *bis*, 1005 (Lep.)
Shannon, J. A., Earle, D. P., Jr., Brodie, B. B., Taggart, J. V., Berliner, R. W. *et al.*, 343 (Mal.)
Shannon, R. C., with Mulrennan & Goodwin, 633 (Mal.)
Shapiro, J. M., Saliternik, Z. & Belferman, S., 247 (Mal.)
Shapiro, S., with Murphy, 58 (Haem.)
Sharp, H. S. & Biggs, R., 1042 (Misc. Dis.)
Shaw, H. C., with Sulzberger & Kanof, 1028 (Der.)
Shear, M. J., with Topping, 457 (Typh.)
Sheldon, W. H., 565 (R.F.)
Shelley, J. E., 487 (Oph.)
Shepherd, H. M. D., 492 (Heat Str.)
Shepherd, W. H. T., 4 (Mal.)
Sher Khan, 323 (Ent.)
Sherlock, S., 1047 (Lab.)
Sherman, W., with Flood, 419 (Reports, etc.)
Shigan Li, with Jettmar, 981 (Typh.)

Shih, F. I., Chang, S. S. & Yuan, K. L., 726 (Pl.)
—— & Pollitzer, R., 725 (Pl.)
Shih Lu Chang, 286 (Am.)
Shircore, J. O., 185 (Bl.)
Shishlaeva-Matova, L. S., 351 (Mal.)
Shkorbatov, V. I., 464 (Typh.)
Shmatikov, M. D., with Krontovskaya, 274 (Typh.)
Shortt, 405 (Ulc.)
Shortt, H. E., 100 (Leish.)
Shrager, J., with Applebaum, 176 (Mal.)
Shubladze, A. K., Soliterman, P. L. & Sterngold, E. J., 461 (Typh.)
Shute, P. G., (434), 630, 857 (Mal.)
Shwachman, H., with Bercovitz & Rodriguez-Molina, 572 (Hel.)
Siddons, L. B., 624, 625 (Mal.)
——, with Das Gupta, 537 (Mal.)
——, with —— & Chakravarti, 177 (Mal.)
Siedek, H., (367) (Typh.)
——, Kasperczik, K. & Fanta, H., 105 (Typh.)
Siegel, H. & Mushett, C. W., 178 (Mal.)
——, —— & Emerson, G. A., 628 (Mal.)
Sieyro Nieto, L., 15 (Tryp.)
Silberstein, J. S. with Sather, 885 (Typh.)
Silva, F., 928 (Der.)
Silva, R. & Castañeda, M. R., 990 (Typh.)
——, with ——, 193, 457 (Typh.)
Silva-Goytia, R., 718 (Typh.)
Silveira, J., 46 (Hel.)
Silveira, L. M., 473 (Lep.)
Silverman, D. N. & Leslie, A., 1001 (Am.)
Silverman, M., Ceithaml, J., Taliaferro, L. G. & Evans, E. A., Jr., 448 (Mal.)
——, with Taliaferro & Coulston, 449 (Mal.)
Simonetti, C., 809 (Am.)
Simpson, M. L., 10 (Mal.)
Sinclair, A. J. M., with Williams & Jackson, 369 (Typh.)
Singal, S. A., with Briggs & Sydenstricker, 821 (Def. Dis.)
Singh, B., 24 (Typh.), 403 (Haem.)
Singh, G., 883 (Typh.)
Singh, I. & Singh, I., 701 (Bl.)
Sinton, J. A., 10 (Bl.), 630, 694 (Mal.), 839 (Ent.)
Skipper, E. W. & Haine, G. L., 451 (Bl.)
Skorin, I. E. & Korshunova, O. S., 638 (Typh.)
Slavina, N. S., 644 (R.F.)
Sliwensky, M., 91 (Mal.)
Sloan, F. R., with Voorhies, 484 (Haem.)
Sloan, N. R., 296 (Lep.)
Slowey, J. F., 148 (Misc. Dis.)
Smadel, J. E., with Hamilton & Plotz, 796 (Typh.)
Smirnov, G. G., 135 (Hel.)
Smith, C. C., with Monro, Briand & Delisle, 759 (Ent.)
Smith, C. N. & Gouck, H. K., 151 (Ent.)
Smith, G. C. & Passalacqua, L. A., 696 (Mal.)
Smith, J. A., with Kean, 338 (Mal.)
Smith, K. E., with Pappenheimer, Thompson & Parker, 826 (Haem.)
Smith, M. I. & Stohlman, E. F., 153 (Ent.)
Smithburn, K. C. & Haddow, A. J., 320 (Misc. Dis.)
Smithburn, K. C. Mahaffy, A. F. & Haddow, A. J., 321 (Misc. Dis.)
Smithers, R. H. N., 404 (Vms.)
Smorodintsev, A. A. & Drobyshevskaya, A. I., 196 (Typh.)
Smyly, H. J., 559 (Am.)
Snow, J. S., 544 (Leish.)
——, Wedding, E. S. & Tomlinson, W. J., (587) (Der.)
Snyder, J. C. & Wheeler, C. M., 987 (Typh.)
——, with Yeomans, Murray, Zarafonetis & Ecke, 201 (Typh.)
Snyder, M., with Plotz, Bennett & Wertman, 636 (Typh.)
Soares, T., 319 (Misc. Dis.)
Soberón y Parra, G., 174 (Mal.)
—— & León y Blanco, F., 1027 (Der.)
Sodeman, W. A. & Lewis, B. O., 562 (Am.)
Soeiro, A., with Foy, Kondi & Rebelo, 634 (Bl.)
Sofia, F., 109 (Typh.)
—— & Spadaro, O., 547 (Typh.)
Sohier, R., Parnet, J. & Chon, A., (795) (Typh.)
Soliterman, P. L., with Shubladze & Sterngold, 461 (Typh.)
Soper, F. L., Wilson, D. B., Lima, S. & Antunes, W. S., 79 (B.R.)
Soricelli, F., (835) (Ulc.)
Sorsby, A., 486 (Oph.)
—— & Hoffa, E., 488 (Oph.)
Souders, C. R., with Jones, 149 (Misc. Dis.)
de Souza, A. R., 567 (Lep.)
de Souza-Araujo, H. C., 132 *bis*, (904), 1006 *bis*, 1007, 1008 *ter*, 1010 *bis* (Lep.)
——, Mariano, J. & de Oliveira Castro, G. M., 1009 (Lep.)
—— & Miranda, R. N., 1007 (Lep.)
Soysa, E. & Jayawardena, M. D. S., 320 (Misc. Dis.)
Spadaro, O., with Sofia, 547 (Typh.)
Speck, J. F. & Evans, E. A., Jr., 967 (Mal.)
Spellberg, M. A., with Browne & McHardy, 729 (Am.)
Spies, T. D., Cogswell, R. C. & Viltner, C., 307 (Def. Dis.)
——, with Moore, Vilter, & Minnich, 482 (Haem.)
Sprague, H. B. & Barnard, J. H., 890 (Y.F.)
Spühler, O. & Kartagener, M., 1039 (Misc. Dis.)
Stabler, R. M., 938 (Prot.)
Stachelek, J., with Pitts, Johnson, Consolazio, Poulin & Razoyk, 226 (Heat Str.)
Stannus, H. S., 10 (Bl.)
Stare, F. J., with Hegsted & McKibbin, 784 (Mal.)
Starling, P., 311 (Der.)
Starovierova, A. G., with Ravikovich, Neuman & Maievsky, 549 (Typh.)
——, with ——, ——, —— & Ratner, 549 (Typh.)
Starr, D. F., 1044 (Ent.)
Stauber, L. A. & van Dyke, H. B., 540 (Mal.)
Stavaux, R., 736 (Ys.)
Stéfanopoulo, G. J. & Duvolon, S., 205 (Y.F.)
—— & Etévé, J., 258 (Tryp.)
Stein, L., with Wertheimer, 262 (Leish.)

Stein, M., with Alexander & Park-Ross, 38 (Am.)
Steinberg, L. D., Kanibolotskaya, E. N. & Karlova, N. S., 339 (Mal.)
Steiner, G. & Steiner, Grete, 74 (Lab.)
Steinhaus, E. A. & Parker, R. R., 375 (Den.)
Steinitz, H., with Klopstock, 590 (Misc. Dis.)
Steinman, H. G., with Eagle, Hogan & Doak, 15 (Tryp.)
Stepanov, I. R., with Gromashevsky, 191 (Typh.)
——, with —— & Bagrova, 460 (Typh.)
Stephens, J. W. W., 541 (Bl.)
Stephenson, C. S., (102) (Typh.)
Stephenson, R. W., 553 (Typh.), 572 (Hel.)
Stephenson, W., 474 (Hel.)
Stern, H. J., with Miterstein, 1030 (Oph.)
Sterngold, E. J., with Shubladze & Soliterman, 461 (Typh.)
Stevens, R. S., 364 (Typh.)
Stevenson, A. C., with Findlay, 349 (Mal.)
Stewart, C. D. & Pilcher, J. F., 712 (Leish.)
Stewart, M. O., Florio, L. & Mugrage, E. R., (119) (Den.)
——, with —— & ——, 118 (Den.)
Stewart, W., 34 (Dys.)
Steyaert, J., with Bessemans, Derom, Doussy & Baert, 870 (Tryp.)
Stockdale, F., 760 (Misc.)
v. Stockert, 459 (Typh.)
Stohlman, E. F., with Smith, 153 (Ent.)
Stohlmann, H., 174 (Mal.)
Stoll, A. M., Ward, P. A. & Mathieson, D. R., 999 (Am.)
Stoll, N. R., with Franks, 916 (Hel.)
Stone, P. H., 70 (Misc. Dis.)
Stowman, K., (719) (Typh.)
Striberny, L., 92 (Mal.)
Strong, R. P., 421 (B.R.)
Stryker, G. V. & Halbeisen, W. A., with Leventhal, L., 581 (Def. Dis.)
Stuart-Harris, C. H., 979 (Typh.)
Stubbs, T. H., 446 (Mal.)
Suárez, J., 813 (Lep.)
Suárez, R. M., 836 (Misc. Dis.)
Sukiennik, S., with Leffkowitz, 591 (Misc. Dis.)
Sulzberger, M. B., Shaw, H. C. & Kanof, A., 1028 (Der.)
Sundaram, S. K., 59 (Haem.)
Suri, H. L., with Andreasen, 742 (Hel.)
Sutton, H., 849 (B.R.)
Swantz, H. E. & Bayliss, M., 701 (Bl.)
Swaroop, S., with Yacob, 620 *bis* (Mal.)
Swellengrebel, N. H., with Schüffner, 922, 924 (Hel.)
Swyer, G. I. M., 1002 (R.F.)
Sydenstricker, V. P., with Briggs & Singal, 821 (Def. Dis.)
Symes, C. B. & Hadaway, A. B., 785 (Mal.)
Symmers, D., 833 (Der.)
Syverton, J. T. & Thomas, L., 879 (Typh.)
Szekely, P., with Hume, 210 (R.F.)
Szucs, M. M., with Zizmor, (477) (Hel.)

T.

Taggart, J. V., with Shannon, Earle, Brodie, Berliner *et al.*, 343 (Mal.)
Tagnon, H. J., 8 (Mal.)
Taliaferro, L. G., Coulston, F. & Silverman, M., 449 (Mal.)
——, with Silverman, Ceithaml & Evans, 448 (Mal.)
——, with Taliaferro, 256 (Mal.)
Taliaferro, W. H. & Taliaferro, L. G., 256 (Mal.)
Talice, R. V., 305 (Hel.)
Tallant, E. J., with Wirts, 206 (Dys.)
Tan, S. C., with Cheng & Liu, 733 (Am.)
Tang, F. F., Chu, C. M. & Wong, Y. W., 120 (Chl.)
Tang, W. K. & Chang, K., 743 (Hel.)
Tanner, F. W., with Severens, 847 (Reports, etc.)
Taubenhaus, L. J., 208 (Am.)
Tatum, A. L., with Gajewski, 92 (Mal.)
Taylor, E. L., 55 (Hel.)
Taylor, F. H., 511 (B.R.)
Taylor, G. F. & Chhuttani, P. N., 750 (Haem.)
Taylor, H. L., with Henschel, Brozek, Mickelsen & Keys, 145 (Heat Str.)
Taylor, M. P., with Fair, Chang & Wineman, 734 (Am.)
Taylor, R., with Fraser, 897 (Am.)
Teixeira, W. G., (1025) (Haem.)
Temkin, O. & Ramsey, E. M., 533 (Mal.)
Theodor, O. H. & Parsons, B. T., 1043 (Ent.)
Thirlby, R. L., (351) (Mal.)
Thomas, C. R., 168 (Rab.)
Thomas, D., with Barrett, 474 (Hel.)
Thomas, H. M., Jr. & Gage, D. P., 1018 (Hel.)
Thomas, L., with Syverton, 879 (Typh.)
Thompson, J. H., 695 (Mal.)
Thompson, P. E., 541, 787 (Mal.)
Thompson, R. B., (1003) (R.F.)
——, with Grant & Anderson, 187 (Tryp.)
Thompson, W. P., with Pappenheimer, Parker & Smith, 826 (Haem.)
Thornton, P. A., 294 (Lep.)
Thurman, D. C., Jr., with Irons, Bohls & McGregor, 462 (Typh.)
Tiant, F. R., 45 (Lep.)
Tiburskaya, N. A., with Sergiev, 622 (Mal.)
Tillman, A. J. B., with Kellner & Hochstein, (964) (Mal.)
Tisdall, F. F., with McCreary, 58 (Def. Dis.)
Tokarevich, K. N., 460 (Typh.)
Tomassi, G., 140 (Def. Dis.)
Tomlinson, W. J., 582, 1025 (Haem.), 938 (Prot.)
—— & Jacob, J. E., 582 (Haem.)
——, with Snow & Wedding, (587) (Der.)
—— & Wilson, L. A., Jr., (592) (Misc. Dis.)
Tompsett, R. R., with Harrison & Barr, 222 (Sp.)
Tong, W. K., with Chang, Chin & Li, 744 (Hel.)
Toomey, A. G. G., 862 (Mal.)
Topping, N. H. & Shear, M. J., 457 (Typh.)
Toranzos, L. B., 874 (Tryp.)
Toro, G., with Patiño-Camargo & de Zulueta, 799 (Typh.)
Torregrosa, M. V., with Oliver-González, 45 (Hel.)
Torres, C. M., (876) (Leish.), (1020) (Hel.)
Torres, U. L., 311 (Der.)
Torres Cañamares, F., (859) (Mal.)
Torrico M., R.A., with Dias, (875) (Tryp.)
Tötterman, G., 215 *quat.* (Hel.)

Tovar, R. M., 721 (Typh.)
Trembley, H. L., with Bishopp, 760 (Ent.)
——, with Coatney & Cooper, 865 (Mal.)
Trespalacios, F., Gonzalez Peris, G. & de Laosa, O., 586 (Der.)
—— & Jover, J., 212 (Lep.)
Treston, M. L., 764 (B.R.)
Treu, R., 410 (Misc. Dis.)
Trevett, L. D., with Hindle, Rose & Prout, 446 (Mal.)
Trincão, C., (1025) (Haem.)
Trinidad Government-Rockefeller Foundation, 617 (Mal.)
Trout, C. L., 900 (Lep.)
Trowell, H. C., 306 (Def. Dis.), 825, 827 (Haem.), 1050 (B.R.)
——, with Brown, 306 Def. Dis.)
Tsuchiya, H. & Reller, H., 817 (Hel.)
Tupínambá, A., with Martins & Versiani, (875) (Tryp.)
Turnbull, H. H., 318 (Misc. Dis.)
Turnbull, R. B., Jr., with Maveety & Bauer, 441 (Mal.)
Turner, R. G., 842 (Ent.)
Tze-Tuan Chen, 185 (Mal.)

U.

Ubatuba, F. & Vieira, G., 996 (Bart.)
Ulitcheva, A. V., 3 *bis*, 4 (Mal.)
Unat, E. K., with Braun, 108 (Typh.)
Underwood, L. J. (710) (Tryp.)
Ungar, H., 745 (Hel.)
Ungar, J., Jr., with Metcalf, 341 (Mal.)
Unsworth, K., 397 (Hel.)
Unti, O., 86 *bis*, 87, 90, 697 (Mal.)
——, with Wasicky, 182 (Mal.)
Ureta, R. E., 225 (Vms.)
Uriguen, D., with Macchiavello, 466 (Pl.)
Usinger, R. L., 542 (Tryp.)
——, with Sabrosky, 244 (Mal.)

V.

Vaccaro, A., with Fernández, Serial & Carboni, 832 (Der.)
Vainberg, V. G. & Gulamova, V. P., with Perekriostova, E. P. & Ostrovskaya, S. G., 549 (Typh.)
Vakil, R. J., 896 (Am.)
Valentine, W. N. & Neel, J. V., 223, (825) (Haem.)
van den Berghe, L., 871 (Tryp.)
—— & Bone, G., (843) (Ent.)
——, with Rodhain, 872 (Tryp.)
van den Ende, M., with Andrewes, King & Walker, 20 (Typh.)
——, with Elford, 455 (Typh.)
——, with Begg & Fulton, 104 (Typh.)
Vanderplank, F. L., 99, (187), 703 *bis* (Tryp.), (231), 760 (Ent.)
——, with Jackson, 187 (Tryp.)
——, with Potts, 869 (Tryp.)
van der Sar, A., 391 (Lep.)
—— & Hartz, H., 837 (Misc. Dis.)
Vander Veer, J. B., with Hodge & Denhoff, 1020 (Hel.)
Varela, G., 364 (Typh.)
——, with Bustamante, 203 (Typh.)
——, with Mariotte & Bustamante, 721 (Typh.)
——, Olarte, J. & Castro Estrada, S., 927 (Der.)
—— & Zozaya, J., (881) (Typh.)
——, with —— & Castro Estrada, 143 (Der.)
Vargas, L., (230), 940 (Ent.), 663 (B.R.), 919 *ter* (Hel.)
Vargas, R., 796 (Typh.)
Vasilkova, Z. G., 569 (Hel.)
Venezuela, Ministerio de Sanidad y Asistencia Social, 868 (Mal.)
Venkat Rao, V., with White, 180 (Mal.)
Venner, R. B., 478 (Hel.)
——, with LeFevre & McDermott, 470 (Ys.)
Venning, W., with Harrell & Wolff, 373, 888 (Typh.)
Versiani, V., with Martins & Tupínambá, (875) (Tryp.)
—— & Renault, L., (209) (Am.)
de Vidas, J., 882 (Typh.)
Videla, C. A. & Scodeller, J. A. (816) (Hel.)
Vieira, G., with Ubatuba, 996 (Bart.)
de Villafane Lastra, T., 892 (Pl.)
Villela, F., with Pessôa, 712 (Leish.)
Villela, G. G. & Linhares, H., (214) (Lep.)
Vilter, R., with Moore, Minnich & Spies, 482 (Haem.)
Viltner, C., with Spies & Cogswell, 307 (Def. Dis.)
Viñes Ibarrola, D. J., 22 (Typh.)
Vinnitzky, I. M., 746 (Hel.)
Vint, F. W., 508 (Reports, etc.)
Visveswar, S. K., with Heilig, 127 (Am.), 442 (Mal.)
Viswanathan, D. K., Rao, T. R. & Rao, T. S. R., 625 (Mal.)
Viswanathan, R., 691 (Mal.)
—— & Natarajan, B., 411 (Misc. Dis.)
Vonfraenkel, P. H. & Krick, E. S., 592 (Misc. Dis.)
Voorhies, N. W. & Sloan, F. R., 484 (Haem.)
de Vries, A., with Braun, (351) (Mal.)

W.

Wagle, P. M., 558 (Pl.)
Wagner, S. M., with Burrows & Mather, 728 (Chl.)
——, with ——, —— & McGann, 727 (Chl.)
Wain, R. L., with Martin, 152 (Ent.)
Walker, C. W., 827 (Vms.)
Walker, J., with Andrewes, King & van den Ende, 20 (Typh.)
Walker-Taylor, P. N., 834 (Ulc.)
Wall, R. & Garvan, J. M., 893 (Dys.)
Wallace, F. G., with Angevine, Hamilton & Hazard, 976 (Leish.)
Walls, L. P., 709 (Tryp.)
Walsh, N., 720 (Typh.)
Walters, W., Watkins, C. H., Butt, H. R. & Marshall, J. M., 37 (Am.)
Walton, A. C., with Wright & Jacobs, 306 (Hel.)
Wanamaker, J. F., (413) (Ent.)

Wang, C. W., with Chung & Lee, (876) (Leish.)
Wanson, M., 412 (Ent.)
—— & Berteaux, M., 337 (Mal.)
——, with Henrard & van Wymeersch, (229) (Ent.)
War Department, Washington, 234 (B.R.)
War Department Technical Bulletin, (864) (Mal.), (876) (Leish.), (896) (Am.)
War Medicine, (893) (Chl.), (896) (Am.), (906), (919) (Hel.), (935) (Ulc.)
War Office, 112 (Typh.), 116 (Y.F.), 692 (Mal.), 742 (Hel.), 999, 1001 (Am.)
Ward, P. A., with Stoll & Mathieson, 999 (Am.)
Ward, R. L. & Mason, A. S., 934 (Ulc.)
Warin, R. P. & Alexander, R. S., 380 (Dys.)
Warshauer, S. E., with Berry & Johnson, 555 (Typh.)
Wartman, W. B., 305 (Hel.)
Washington, 570 (Hel.)
Wasicky, R. & Unti, O., 182 (Mal.)
Waterlow, J. C., with Ladell & Hudson, 143 (Heat Str.)
Waterman, J. A., (439) (Mal.)
Watkins, C. H., with Butt, Hall & Cragg, 783 (Mal.)
——, with Walters, Butt & Marshall, 37 (Am.)
Watson, R. B. & Rice, M. E., 173 (Mal.)
Wedd, G., with Carter & d'Abrera, 73 (Ent.)
Wedd, S., 111 (Typh.)
Wedding, E. S., with Snow & Tomlinson, (587) (Der.)
Weiner, J. S., 587 (Heat Str.)
Weinman, D., 423 (B.R.)
——, with Augustine & McAllister, 288 (R.F.)
Weiss, J. E., with Finerman, 284 (Dys.)
Welch, P. B., 287 (Am.)
Weller, T. H., with Dammin, 941 (Lab.)
Wellman, W. E., with Englehorn, 819 (Hel.)
Wells, A. H., with Hatch, 312 (Der.)
Wenderoth, H., 482 (Hel.)
Wenrich, D. H., 386 (Am.)
Wenyon, C. M., 839 (Ent.)
Wertheimer, E. & Stein, L., 262 (Leish.)
Wertman, K., 635 (Typh.)
——, with Plotz, Bennett & Snyder, 636 (Typh.)
Wessels, A. L., with Cooper, 857 (Mal.)
West, J. B. & Henderson, A. B., 253 (Mal.)
West, T. F., with Campbell, 152 (Ent.)
Westfall, B. A., with Ellis & Ellis, 256 (Mal.)
Wexberg, L. E., with Applebaum, 816 (Hel.)
Wharton, L. H., 737 (Lep.)
Wheeler, C. M., with Snyder, 987 (Typh.)
Wheeler, W. E., 811 (R.F.)
White, R. Senior & Venkat Rao, V., 180 (Mal.)
Whitehill, R. & Austrian, R., 470 *bis* (Ys.)
—— & Miller, M. H., 576 (Hel.)
Whitelaw, J. C. G. & Sandy, F., (304) (Hel.)
Whittier, L., with Einhorn & Miller, 911, 912, 914 (Hel.)
Whittingham, H., 255 (Mal.)
Wickramasinghe, W. G., (1049) (Reports, etc.)
Wigglesworth, V. B., 255 (Mal.)
Wilbar, C. L., Jr., with Witlin, 642 (Pl.)
Wilckens, H., 266 (Typh.)
Wilder, R. M., Jr., (222) (Sp.)
Wilkinson, P. B., 137 (Def. Dis.)
Williams, C. L., 117 (Y.F.)
Williams, E. R. & Davies, A. B., (404) (Vms.)
Williams, R. W., 367 (Typh.)
Williams, S. W., Sinclair, A. J. M. & Jackson, A. V., 369 (Typh.)
Williams, W. L., 14 (Tryp.)
Willmore, J. G., 559 (Am.)
Wilson, C. S., Mathieson, D. R. & Jachowski, L. A., 73 (Ent.)
Wilson, D. B., with Annecke & McCarthy, 966 (Mal.)
——, with Soper, Lima & Antunes, 79 (B.R.)
Wilson, G. S., 843 (Lab.)
Wilson, H. A., with Marsh, 588 (Ulc.)
Wilson, L. A., Jr., with Tomlinson, (592) (Misc. Dis.)
Wilson, R. P., with Bland, 1031 (Oph.)
Winblad, F. A. S., (420) (Reports, etc.)
Wineman, M. A., with Fair, Chang & Taylor, 734 (Am.)
Winfield, G. F. with Ch'ing Hua Meng, (842 *bis*) (Ent.)
Winkle, S., 545, 546 (Typh.)
Winkler, A. W., with Elkinton, 316 (Heat Str.)
Winsor, T. & Burch, G. E., 484, 1024 (Haem.), 752 (Heat Str.)
——, with ——, 752 *bis* (Heat Str.)
Wirts, C. W., Jr. & Tallant, E. J., 206 (Dys.)
Witenberg, G., 396 (Hel.)
Witlin, B., 232 (Lab.)
—— & Wilbar, C. L., Jr., 642 (Pl.)
Wolff, W. A., with Harrell & Venning, 373, 888 (Typh.)
Wolfson, F., 867 (Mal.)
Wolman, M., 469 (R.F.)
Wolpers, C., 85 (Mal.)
Wolstenholme, B. & Gear, J., 877 (Typh.)
Womersley, H., 324 (Ent.)
—— & Heaslip, W. G., 28 (Typh.), 324 (Ent.)
Wong, Y. W., with Tang & Chu, 120 (Chl.)
Wood, P., 250 (Mal.)
Wood, S. F., 710 *bis* (Tryp.)
Woodhill, A. R., with Lee, 1049 (B.R.)
Woods, R. P., with Burchenal, 635 (Leish.)
Woodward, G. E., 281 (Pl.)
Woodward, T. E., with Blanc, 547 (Typh.)
——, with Bland, E. F., 199 (Typh.)
Worth, C. B., with Mackie & Hunter, 665 (B.R.)
Wright, C. I. & Sabine, J. C., 252 (Mal.)
Wright, D. O. & Gold, E. M., 1041 (Misc. Dis.)
Wright, F. J., 498, (659) (Misc. Dis.)
—— & Roberts, J. I., 215 (Hel.)
Wright, W. H., with Cram & Jones, 646 (Hel.)
——, Jacobs, L. & Walton, A. C., 306 (Hel.)
Wu, C. C., 39 (Am.)
—— & Chi, C. Y., 795 (Typh.)
Wyckoff, R. W. G., with Donovick, 792, 882 (Typh.)
van Wymeersch, H., with Henrard & Wanson (229) (Ent.)
Wyne, P. S., with Jacoby, Loudon & Failmezger, 559 (Dys.)

Y.

Yacob, M., 119 (Chl.)
—— & Swaroop, S., 620 *bis* (Mal.)
Yarygin, N. E. & Nagibina, N. I., 349 (Mal.), 989 (Typh.) ;
Yates, W. W., with Knipling & Gjullin, 73 (Ent.)
Yeomans, A., Snyder, J. C., Murray, E. S., Zarafonetis, C. J. D. & Ecke, R. S., 201 (Typh.)
Yoeli, M., with Measham & Finnegan, 695 (Mal.)
Yolles, S. F., with Yolles & Byrd, 437 (Mal.)
Yolles, T. K., Yolles, S. F. & Byrd, D. A., 437 (Mal.)
Young, E. A. E., with Harington, 271 (Typh.)
Young, E. G., & Rice, F. A. H., 415 (Lab.)
Young, M. D., 686 (Mal.)
——, with Burgess, 437 (Mal.)
——, with Ehrman & Ellis, 628 (Mal.)
Young, R. M., with Schnelle, 52 (Hel.)
Young, W. A., 211 (Ys.)
Yu, N. G. & Mao, C. P., 747 (Hel.)
Yuan, K. L., with Shih & Chang, 726 (Pl.)
Yudkin, J., 596 (Reports, etc.), 863 (Mal.)

Z.

Zair, A. H., 111 (Typh.)
Zambra, E. R., 17 (Tryp.)
Zanetti, V., 741 (Lep.)
Zarafonetis, C. J. D., with Parsons, 654 (Der.)
——, with Yeomans, Snyder, Murray & Ecke, 201 (Typh.)
Zia, S. H., with Chen & Anderson, 734 (R.F.)
Zizmor, J. & Szucs, M. M., (477) (Hel.)
Zozaya, J., with Varela, (881) (Typh.)
——, —— & Castro Estrada, S., 143 (Der.)
Zuckerman, S. S. & Hibbard, J. S., 480 (Hel.)
Zuelzer, W. W., 71 (Prot.)
de Zulueta, J., with Patiño-Camargo & Toro, 799 (Typh.)
Zumpt, F., 505 (Ent.)
Zwemer, R. L., with Kessler, 889 (Bart.)

INDEX OF SUBJECTS.

(The entries in heavy type refer to Sections in which abstracts on the subject indicated are grouped together. Page numbers within brackets indicate papers cited by title only.)

Abscesses, "filarial", 497
Acanthocheilonema perstans infection, in Nigeria, 649
Acariasis, pulmonary
in Singalese soldiers, 320
and asthma, relationship between, 320
diagnosis, differential, from
bronchomycosis, 320
cutaneous disease, 320
helminthiasis, 320
leukaemia, eosinophilic, 320
pneumoconiosis, 320
tuberculosis, 320
in monkeys, 595
symptoms, 320
treatment
carbarsone, 320
stovarsol, 320
Actinomyces mexicanus infection, treatment by sodium propionate, failure of, 142
Actinomycosis of urinary bladder, complicating madura foot, 312
Adenolymphocele
in Belgian Congo, 820
inguinal glands in, 820
Aëdes spp., 503
in Venezuela, 323
abnormalis, virus, 320, 321
aegypti
in U.S.A., Texas, variable prevalence of, 757
Anti-, Measures, Organization of Permanent Nation-wide, in Brazil (book review), 79
breeding
habits of, 938
method employed in, for biological assay of insecticides, 229
control
in U.S.A., 374
larvicides, phenothiazine, 32
thiamin chloride as repellent, failure of, 73
larvae, nutrition of, (1043)
hensilli n.sp., 504
samoana, habits of, 479
scutellaris
vector of
dengue, in New Guinea, 656
filariasis, 53
hebrideus, vector of dengue in New Hebrides, 32
horrescens, breeding habits of, 938
pseudoscutellaris, habits of, 479, 938
taeniorrhynchus, experimentally infected with *W. bancrofti*, 917
African Sick. A Clinical Study [GELFAND] (book review), 78
Agamofilaria streptocerca, in Belgian Congo, 821
Aircraft, disinfection of, in yellow fever control, 557
Allergy, as cause of pemphigus, 311
Alopecia, leprotic, in Mexico, (1010)
Altitude, heart disease and, 67
Amblyomma spp.
control by spraying, 151
in dogs, in China, 742
cajennense
in Venezuela, 323
experimentally infected with *Myco. lepra murium*, (904)
Amidines, aromatic, estimation of, 1048
AMOEBIASIS AND INTESTINAL PROTOZOAL INFECTIONS, 36–41, 124–13(207–209, 285–288, 381–38(468, 559–564, 643–644, 729 734, 809–810, 894–897, 999 1002, *see also Entamoeba Hi tolytica*
Amoebiasis, (969)
in Africa, 410
East, 1038
Angola, 319
Argentine, 37, 731
Asia, South-East, 753
Australia, 835
China, 39
India, 381, 408, 731
Marshall Islands, 420
Mexico, 895
in children, 36, 594
Middle East, 147, 1002
New Guinea, 808
New Zealand, in naval personnel, 468
Pacific area, 319, 657
Palestine, 128, 894
Solomon Islands, 657
South Africa, 38, 643, 894
in infants, 285
U.S.A., 208, 379, 729
in children, 644
of anus and perineum, in India, 730
as cause of recurrent abdominal pain, 208
in children, 36, 285, 594, 644
chronic
treatment, 128, 561
in troops, 384
complications, 729
brain abscess, 126
hepatitis, 319, 382
intussusception, (286)
liver abscess, 319, 382
cutaneous, 38
diagnosis, 319, 383, 384, 594, 729
differential, 125
difficulties of, 124
gastric function in, 643
hepatic, 562
hepatitis, 319, 382
in infants, 285

Amoebiasis—*cont.*
influence of sex in, 895
iron-deficiency, in Indian soldiers, 651
liver
abscess, 319, 382
treatment, 1001
penicillin, (810)
unsuspected, 37
function in, 127
perinephric abscess, 38
in returned soldiers, 319
treatment (including experimental), 319, 383, 384, 559, 729, 809, 895, 999
alkaloids of *Holarrhena antidysenterica*, pharmacological tests of, 39
carbarsone, 126
comparative values of, 207, 563
diodoquin, 731
sequelae, 1001
emetine, 37
variations in liver function during, 127
enterovioform, sequelae, 896
iodochlorhydroxy-quinoline, trials of, 286
shadocol, 38
sulphonamides, 753
succinyl sulphathiazole, 1002
sulphaguanidine and sulphathiazole, 1002
ya tan tzu, 39
toxicology of, 733
tuberculosis and, 731
Urinary (A Critical Review) (special article), 947–959
urticaria and, 37
of uterus, 38
variability of, 730
water treatment and, (896)
amoebic
abscess of lung, (999)
colitis, epigastric, and appendicular syndrome in, 809
cystitis, treatment by sulphonamide, 209
typhlitis, 382
anaemia
in Asia, South-East, in Indian troops, 754
Assam tea estates, 233
India, 407
Indian troops, 754, 822
aetiology, 308
associated with unidentified erythrocytic inclusions, 826
bartonella, experimental, 889
classification, 308
Cooley's
in Egyptian children, 224
India, 59
U.S.A., 223
target cells in, (825)
transmission, 223
haemolytic
caused by plasmoquine and quinine, (353)
congenital, in negroes, 583
macrocytic
in India, 222
Persia and Iraq Force, in Indian soldiers, 403
as aid in diagnosis of deficiency dermatoses, 581
nutritional
in India, 59
Uganda, 827
Anaemia—*cont.*
macrocytic—*cont.*
nutritional—*cont.*
in U.S.A., 482
treatment, 483
dehydrated liver, 827
refined liver extracts, 59
vegetarianism and, 750
treatment, 403
malarial, 338
Mediterranean, 484
sickle-cell, 484
in Angola, (1025)
Argentine, (141)
Bantus, 825
Honduras, in Carib Indians, 141
Panama, 582
St. Thomas Island, (1025)
South Africa, 652
abdominal crisis in, 1025
characteristic habitus of patients with, 1024
diagnosis, 825
physiochemical blood test, 484
erythrocytes, behaviour of, in, 58
polyarthritis and, 141
terminology, 308
Anaemias, Infectious, due to Bartonella and Related Red Cell Parasites [WEINMAN] (book review), 423
Ancyclostoma duodenale see under Hookworms
Ankylostomiasis
in Africa, East, 1038
Angola, 319
Australia, 835
China, 743
Eritrea, 478
Kenya, 48
New Guinea, 743
New Zealand, in naval personnel, 468
Pacific area, 49, 319, 658
Panama, in children, 911, 912
Solomon Islands, 658
South Africa, 894
anaemia, urinary excretion of sodium chloride in, 1020
complicating other diseases, 742
in dogs, in China, 742
eosinophilia and, 49
geophagy and, 943
Necator americanus infection
in China, 743
New Guinea, 743
Northern Rhodesia, 48
pulmonary infiltrations in, 592
symptoms, fever, 48
treatment by carbon tetrachloride, 48
in returned soldiers, 319
symptoms, 743
atypical, 48
treatment
carbon tetrachloride, 49, 176
chenopodium oil, 49
gentian violet, 912
tetrachlorethylene, 911
and oil of chenopodium, 319, 743
Anopheles
of Africa, West, 434
Algeria, 698
America, North and South, 87

Anopheles—*cont.*
of Arabia, 335
Belgian Congo, 172
Yangambi, (688)
Belgium, (778)
Brazil
Island of Santo Amaro, (173)
São Paulo, 87
British Guiana, 615, 616, 785
Ceylon, 337
Far East, 623
Gold Coast, 255
India, 85, 180, 618, 619, 623
Iran, 248, 533
Kenya, 959
Melanesia, (778)
Pacific area, 319
Palestine, 247
Spain, 171, (859)
Sudan, 1042
Switzerland, 685
Trinidad, 617
U.S.S.R., 254
U.S.A., 173
Pacific coast, 437
Venezuela, 322
adult, sterile, rearing of, (229)
anabasine sulphate as protection against bites of, 1045
attacks, activity in, 230
breeding waters of, in São Paulo, iron content of, 87
Concerned with Transmission of Disease in Man, List of (special article), 517–525
control, 939
in America, North and South, 87
Argentine, (448)
British Guiana, 616
Greece, 447
India, 619
Kenya, 447
Trinidad, 618
U.S.A., 446
anti-larval siphons, (448)
D.D.T., 182, 537, 757, 785
thanite sprays, 632
dissolved oxygen in water of breeding places, 86
drainage, 246
swamp, 786
dusting, pyrethrum, 447
engineering work, 619
fish breeding, 92, 246, 619
in India, (967)
Punjab, comparative value of, (92)
on impounded waters, relation of plants to, 626
larvicides, 1044
D.D.T., 182, 537, 757, 785
mineral oils emulsions, 632
oil-emulsion, 73
Paris green, 246, 351, 619
use of explosives for application of, 4
oiling, 92, 246, 619
spraying, 92, 180, 255, 619
trapping, 72, (940)
exotic, importation of, into U.S.A., 633
flight and dispersion habits, of, 435
Indian, identification of, 85

Anopheles—*cont.*
larvae
and pupae, survival of, in mud, 626
in rice fields, ecology of, 4
population, fluctuation in density of, 858
sporozoites, number found in salivary glands 857
sub-genus *Kerteszia*, rôle of, in malaria, 688
Anopheles
aconitus
eggs of, 337
habits of, 624
and malaria, 623
albimanus
in Venezuela, 250
flight range of, 436
albitarsis
breeding places of, 87
eggs, resistance of, 86
habits of, 615
albitarsis domesticus, rearing of, in laboratory, 4
algeriensis
in Spain, (859)
biology of, 859
amictus, habits of, 624
annularis
eggs of, 337
habits of, 624
and malaria, 623
annulipes, habits of, 624
antunesi, breeding places of, 87
aquasalis
habits of, 617
and malaria, in Trinidad, 617
argyritarsis
breeding places of, 87
and malaria, in Grenada, 86
rearing of, in laboratory, 4
baezai, habits of, 624
bancrofti
habits of, 624
and malaria, 623
barbirostris
eggs of, 337
habits of, 624
and malaria, 623
oviposition of, 337
bellator
breeding of, 4
characteristics of, 688
and malaria, in Trinidad, 617
bifurcatus, and malaria, in Switzerland, 685
boliviensis, characteristics of, 688
brunnipes, infectivity of, 337
cruzi, characteristics of, 688
culicifacies
eggs of, 337
flight, range of, 436, 687
habits of, 624
infectivity of, influence of temperature a humidity on, 625
and malaria, 623
in Arabia, 335
Ceylon, 684
India, 619
darlingi
in Venezuela, 250
breeding places of, 87
flight range of, 436

Anopheles—cont.
darlingi—cont.
habits of, 615, 616
and malaria, in British Guiana, 614, 616, 785
dureni, larva and nymph of, (229)
emilianus, biology of, 961
flavicosta in West Africa, 435
fluviatilis
control by spraying, 180
flight range of, 436
habits of, 619, 624, 625
and malaria, 623
in India, 619
funestus, flight range of, 436
gambiae
in French West Africa, 247
Sudan, 246
and *Anopheles melas*, difference between, 3
biology of, 247
breeding
habits of, 246
at high altitudes, 959
temperature for, 959
ecology, 960
eggs, viability of, away from water, 960
flight range of, 436
gametocyte threshold for infection of, 860
and malaria
in Arabia, 335
Kenya, 959
outdoor resting places of, 336
var. *melas*
biology of, 860
gametocyte threshold for infection of, 860
influence of climate on population of, 245
gigas var. *refutans*, eggs of, 337
hyrcanus
and malaria, in Iran, 249
habits of, 624
and malaria, 623
var. *nigerrimus*
eggs of, 337
habits of, 624
and malaria, 623
var. *sinensis*
habits of, 624
and malaria, 623
implexus, in West Africa, 435
jamesi, eggs of, 337
jeyporiensis
habits of, 624
var. *candidiensis*
habits of, 624
and malaria, 623
karwari
eggs of, 337
habits of, 624
kochi
habits of, 624
and malaria, 623
lanei, breeding places of, 87
laneanus n. sp., (72)
leesoni, in West Africa, 435
leucosphyrus
eggs of, 337
habits of, 624
and malaria, 623
lutzi
breeding places of, 87
characteristics of, 688
Anopheles—cont.
maculatus, eggs of, 337
flight range of, 436
habits of, 624
and malaria, 623
maculipennis
flight range of, 436
and malaria
in Finland, 622
Iran, 249, 533
race *atroparvus*
coexisting with race *labranchiae*, (859)
flight range of, 436
race *freeborni*, feeding habits of, 437
race *labranchiae*
coexisting with race *atroparvus*, (859)
laboratory fertilization of, 173
race *messeae*, in Mongolia, 249
race *sacharovi*, larval distribution of, 3
race *typicus*, and malaria, in Switzerland, 685
mangyanus, habits of, 624
marshalli, in West Africa, 435
melas and *Anopheles gambiae*, differences between, 3
minimus
in Burma, 180
China, 180
Indo-China, 180
control by spraying, 180
flight range of, 436
habits of, 624
and malaria, 623
in Indo-China, 336
var. *flavirostris*
flight range of, 436
habits of, 624
and malaria, 623
moucheti, biology of, (688)
multicolor, flight range of, 436
noroestensis
breeding places of, 87
eggs, resistance of, 86
novumbrosus
habits of, 624
and malaria, 623
obscurus var. *nowlini*, in West Africa, 435
pallidus
eggs of, 337
habits of, 624
parvus, breeding places of, 87
pattoni
habits of, 624
and malaria, 623
pessoai
in Trinidad, 437
breeding places of, 87
pharoensis, laboratory rearing of, 1043
philippinensis
habits of, 624, 626
and malaria, 623
in India, Bengal Delta, 625
pseudopunctipennis
in Grenada, 86
feeding habits of, 437
flight range of, 436
pulcherrimus, habits of, 624
punctipennis, feeding habits of, 437
punctulatus
habits of, 624
and malaria, 623

Anopheles—cont.
Punctulatus—cont.
var. *farauti*, ? synonym of *A. punctulatus moluccensis*, 688
var. *moluccensis*, 778
habits of, 624
and malaria, 623
? synonym of *A. punctulatus farauti*, 688
quadrimaculatus
in U.S.A., Wisconsin, 433
breeding habits of, 626
control
in U.S.A., North Carolina, 537
airplane dusting with Paris green for, 184
feeding habits of, 437
flight range of, 436, 687
and malaria, in U.S.A., 626
production of, relation of intersection line in, 182
seasonal variations of, 687
ramsayi, habits of, 624
rivulorum, in West Africa, 435
saccharovi
flight range of, 436
outdoor resting places of, 336
separatus, habits of, 624
sergenti
flight range of, 436
habits of, 248
outdoor resting places of, 336
splendidus, habits of, 624
stephensi
flight range of, 436
habits of, 624
and malaria, 623
in Arabia, 335
var. *mysoriensis*
habits of, 624
and malaria, 623
strodei
breeding places of, 87
eggs, resistance of, 86
subpictus
eggs of, 337
habits of, 624
oviposition of, 337
var. *indefinitus*
habits of, 624
malaria and, 623
sundaicus
flight range of, 436
habits of, 624
and malaria, 623
superpictus
flight range of, 436
habits of, 624
and malaria, 623
outdoor resting places of, 336
tarsimaculatus
biology of, 961
habits of, 615
laboratory rearing of, 4
tesselatus, eggs of, 337
habits of, 624
and malaria, 623
theileri var. *brohieri* in West Africa, 435
triannulatus, habits of, 615
umbrosus
flight range of, 436
habits of, 624

Anopheles—cont.
and malaria, 623
vagus
eggs of, 337
habits of, 624
varuna
control by spraying, 180
eggs of, 337
habits of, 624
and malaria, 623
oviposition of, 337
walkeri
in U.S.A., (627)
in diurnal shelters, (627)
wellcomei in West Africa, 435
Anopheline Mosquitoes of Australasian Regio (book review), 1049
Anoxemia, influence of, on haemopoietic activit 824
Anthelmintics
acranil, 909
aspidium, 570
carbon tetrachloride, 48, 49, 176, 570
chenopodium oil, 49, 319, 570, 743, 914
gentian violet, 135, 577, 578, 911, 912, 91 921, (1021)
hexylresorcinol, 299, 577, 911, 914
Leche de Higueron, failure of, 912
male fern, 573
methylene blue, 400
pelletierine, 570
pepo, 570
quassia, 578
santonin, 570
spigelia, 570
synthetic lactones and compounds allied santonin, 571
tetrachlorethylene, 299, 319, 570, 578, 74 911, 914
thymol, 570
triphenylmethane derivatives (1021(
ANTIVENENES AND VENOMS, 62–65, 141–14 224–225, 309–310, 403–40 583–584, 652–654, 827–82 1026
Antivenenes
anavenins in production of, (584)
bee, trials of, 405
scorpion, 404
reinforced with physiological saline, 141
snake
Bitis, 829
Cerastes, 404
cobra, potency of, 830
Naja, 829
Sepedon, 829
spider, *Latrodectus mactans*, 65
in treatment of blackwater fever, 701
Trimeresurus barnetti, 65
Anuria, traumatic, 452
fluid intake in, 788
Appendicitis
in East African native, 71
diagnosis, differential, from bacillary dyse tery, 997
malaria and, (964)
oxyuriasis and, 137
verminous, rôle of helminths in, 473
Appendix, containing proglottis of Taenia s pathological changes in, (102

Argasidae of North and Central America and Cuba (book review), 154
Ariboflavinosis
in China, 650, 651
Peru, 927
U.S.A., 482
causing eye diseases, 1036
relationship of, to corneal vascularization, 58
symptoms, 927
Arsenicals, poisoning by, histology of, 595
Arthropods
diseases transmitted by, in Texas, (229)
Vertebrates and, examined in Investigations of the Epidemiology of Yellow Fever (special article), 597–609
Ascariasis
in Africa, East, 1038
China, 744
Morocco, in children, 420
New Zealand, in naval personnel, 468
Palestine, 745
Panama, in children, 911, 914
South Africa, 894
U.S.A., in children, 644
of bile ducts, 51
in children, 420, 644, 744, 911, 914
complications due to, 745
Loeffler's syndrome and, 1039, 1040
transmission, pickled vegetables, 744
treatment
chenopodium oil, 914
hexylresorcinol, 911, 914
tetrachorethylene, 914
Ascaris
causing
cholangitis, 304
pancreatitis, 304
eggs, flies as carriers of, 759
Ascaris
lumbricoides
causing intestinal obstruction, (915)
eggs, effect of sludge digestion on, 818
histological reaction of experimental animals when parenterally introduced, 746
perienteric fluid of, (577)
polysaccharide fraction isolated from, 45
suis
oxidative metabolism of, (219)
polysaccharide fraction isolated from, 45
Asthma and pulmonary acariasis, relationship between, 320
Australorbis centrimetralis
control
copper sulphate, 1018
slaked lime, 1018
vector of schistosomiasis, in Brazil, 1018
Bacteria, inheritance of environmentally induced characters in, 847
Bacterium dysenteriae
transmission, by flies, 35
viability of, 35
Balantidium coli infection, in Morocco, in children 420
Banti's syndrome due to schistosomiasis, 646, 815
Bartonella, Infectious Anaemias due to, and Related Red Cell Parasites [Weinman] (book review), 423
BARTONELLOSIS, 889, 996
in Colombia, 889
experimental
anaemia in, 889
treatment, penicillin, 996
Behcet's complex, (66)
Beriberi
in Angola, 319, 320
Hong Kong, 138
Marshall Islands, 420
U.S.A., 482
cardiovascular, 55
treatment, 56
treatment
Benerva, 320
Betaxan, 320
Bile pigments, detection of, in urine, 596
Bilharzia, *and see* Schistosomiasis
Control of, in Southern Rhodesia [Mozley] (book review), 516
BLACKWATER FEVER, 10–13, 95, 185–186, 257, 353–354, 451–452, 541, 634, 700–702, 788, 968
in Africa, West, 10, 12, 451
Greece, 92
India, 701
Tanganyika, 185
South Africa, (95)
U.S.S.R., 257
in African girl, 541
anuria, 10
blood agglutinins in, 353
circulation, survival of red cells in, 634
fluid intake after haemolytic transfusion reaction, 788
haemoglobinuria, 12, 701
following plasmoquine therapy, (351)
paroxysmal, paralytic
in Sweden, 60
blood in, 61
in typhoid fever, (659)
haemolysis, mechanism of, 452
malaria and, 409, 451, 701
oliguria in, 12
pathogenesis, 12
pathology of, (354)
red cells, survival of, in normal circulation, 634
Ryle's stomach tube in, 146
treatment, 13, 92
alkali, dangers of, 10, 12
antivenene, 701
blood transfusion, 257
sodium bicarbonate and perchloride of mercury, 185
vascular collapse in, 95
Blastomycosis, *see also* Chromoblastomycosis
in Argentine, 143
classification of, 143
cutaneous, in U.S.A., (67)
pulmonary, in brothers, (143)
treatment, 312
Blindness
in Cyprus, 487
South Africa
from eating *Rhodomyrtus macrocarpa*, 68
quinine, 91
Blood
agglutinins, in blackwater fever, 353
chemistry of tsutsugamushi disease, 885
eosinophilia, caused by mepacrine therapy, 630

Blood—*cont.*
erythrocyte
mammalian, diameters of, 187
sedimentation rate in
hepatitis, infectious, 250
malaria, 250
sickle-shaped, in normal saline, plasma and sera, 582
films, staining of, 414
haemoglobin
determination of, variations in, 1023
estimation of, photoelectric, 1023
in haemoglobinuria, paroxysmal, paralytic, 61
haemolysis, mechanism of, in blackwater fever, 452
in malaria, sensitivity of, to haemolysis, 700
normal standard, in Witwatersrand, 824
picture
in dengue, 206
in favism, 61
of *Macacus rhesus*, (141)
in schistosomiasis, 1016
asymptomatic, 572
in trypanosomiasis, 15
in typhus, 204
mite-borne, 111, 370
plasma
determination of mepacrine in, 342
transfusions, in treatment of leprosy, 297
pressure, in Panama, in Cuna Indians, 420
proteins of African negroes, 845
quinine
estimation of, in, 90
and quinidine content of, estimation of, 697
reticulocytes in malarial anaemia, 338
studies, at high altitude, 824
transfusion
and malaria transmission, 174, 627
control of, 688
in treatment of blackwater fever, 257
and typhus transmission, 988
BOOK REVIEWS, 78–80, 154, 234, 327–328, 421–428, 511–516, 663–666, 763–766, 849–850, 944–946, 1049–1052
Borrelia gallinarum, cultivation, 735
Boutonneuse fever, *see under* **TYPHUS GROUP OF FEVERS**
British Empire Leprosy Relief Association (Madras), annual report 1943–1944, 290
Bronchomycosis, diagnosis, differential, from pulmonary acariasis, 320
Bronchopneumonia, in Belgian Congo, 836
Bulinus spp.
in Belgian Congo, (815)
control, 1017
Bulinus
forskalii, in South Africa, 815
truncatus
growth, 1017
life-span, 1017
Bullis fever, *see under* **TYPHUS GROUP OF FEVERS**
Bung-Pagga's disease *see* Myositis, tropical
Cancer
in Cameroons, 936
Ceylon, 149
of liver
in Dutch West Indies, 592
Cancer—*cont.*
of liver—*cont.*
associated with schistosomiasis, 592
clinical features of, 321
in negroes, 150
of oesophagus, in British West Indies, (592)
Cancrum oris *see* Noma
Carate in Venezuela, 323
Carcinoma pain, treatment by cobra venom, (65
Cardiaco negro syndrome, 143
Catha edulis
causing insanity, 658
cultivation, control of, 659
poisoning, 658
Cephalosporium sp. infection, treatment b sodium propionate, failure o 142
Ceratopogonidae, 940
Chagas's disease *see* **TRYPANOSOMIASIS,** human, American
Cheilitis
in Marshall Islands, 420
actinica, in Turkey, 310
Chilomastix mesnili infection,
in East Africa, 1038
Morocco, in children, 420
U.S.A., in children, 644
Cholangitis caused by Ascaris, 304
Cholecystitis, malaria resembling, (862)
CHOLERA, 34, 119–121, 282–284, 377–379, 46 643, 727–728, 806–808, 89 893
Cholera, (893)
in China, 1048
India, 119, 120, 282, 643, 892
Asiatic, giardial dysentery simulating, 129
climate and, 282
control, inoculation of pilgrims, 282
Summary of Recent Abstracts, 1–2
toxin, 807
preparation of, 34
transmission, 283
treatment (including experimental), 120
extract of suprarenal cortex, 643
intravenous transfusions,
saline, pyrogenic reactions following, 37
technique of, 283
mepacrine, 892
sulphaguanidine, 643
sulphathiazole, 643
vaccination, 283, 284
vaccine, mouse protection test for, 467
vibrios
El Tor, lecithinase activity of, 283
endotoxin of, 727, 728
V. cholerae
bacteriophage, synergistic effect of s phanilyl benzamide, 893
biochemistry of, 377, 378
bionomics, 283
isolated from Kunming epidemic, 120
lecithinase activity of, 283
starch digestion, 806
Chromoblastomycosis, *see also* Blastomycosis
in Panama, (587)
experimental, 833
Chrysomia
inclinata, transmitting myiasis, 230
megacephala, life history of, (842)
Cimex hemipterus, in Venezuela, 323

Cinchonidine, distribution of, in fluids and tissues of dogs, 778
Cinchonine, distribution of, in fluids and tissues of dogs, 778
Climate
  effect of, on radiological equipment, 848
  hot
    diuretic response of man in, 587
    effect of, on Europeans, 143
    physiology and, 751
    work and, 145
  human comfort and, 928
  humid, adaptation to changes in, 930
  mountain and coastal, effect of, on nervous system, (327)
  relation of, to
    cholera incidence, 282
    skin diseases, 310
Clinostoma complanatum infection, 396
Clonorchiasis in Pacific area, 319
Coccidioidomycosis
    in U.S.A., 66, 225, 485, 655
      U.S. air personnel, 313
  diagnosis, 225, 485
  primary, possible pediatric problem, (655)
  treatment, 485
    penicillin, failure of, 66
Cold agglutination test, 495
Colitis, ulcerative, treatment, 896
Colonies, British, Welfare in (Mair) (book review), 944
Colorado tick-fever
  and dengue, diagnosis, differential, 118
  symptoms, 117
  transmission, experimental, 118
Configuration and antiplasmodial activity, (91)
Conjunctivitis
    in Middle East, 1037
      Near East, 1030
  treatment
    penicillin, 1036
    sulphonamides, 1030, 1037
  vernalis
    in Palestine, 1031
    treatment, 1032
Corneal vascularization, relationship of riboflavin deficiency to, 58
Creeping eruption, Loeffler's syndrome and, 1041
Cricetus auratus, haematological findings in, (119)
Cienocephalus felis
  in dogs, in China, 742
  rôle of, in transmission of endemic typhus, 462
Culex
  erythrothorax, experimentally infected with *W. bancrofti*, 917
  fatigans
    breeding habits of, 938
    experimentally infected with *W. bancrofti*, 917
  fuscanus, susceptibility of, to infection with *Microfilaria malayi*, 918
Culicidae
  of France, (594)
    Portugal, (1043)
    Sudan, 1042
    Venezuela, 465, 640
Culicines n.sp., in Abyssinia, (940)
Culicoides, 940
Cymtopogon citratus, dermatitis caused by, 66
Cysticercosis
    in Germany, 134
      Mexico, 647
      U.S.A., 816, 907
  cerebral, in Chile, 908
  diagnosis, 134
  pathology, 134
  symptoms, 134
  treatment, 134
Cysticercus cellulosae, single and multiple, in muscle, 134
Cystitis, amoebic, treatment by sulphonamide, 209
Datura poisoning
  in Eritrea, 498
    Tanganyika, 498
    Uganda, 497
DEFICIENCY DISEASES, 55–58, 137–141, 306–308, 580–582, 650–651, 748–749, 821–822, 926–927, *see also under* individual diseases
Dehydration
  in man, 494
  treatment, Ryle's stomach tube, 146
  in troops, in South-East Asia, 754
DENGUE, 32, 117–119, 205–206, 374–375, 641–642, 803–804, 891–892, 996–997
    in Cuba, 803
      Hawaii, 641, 891
      India, 409
      Marshall Islands, 420
      Middle East, 147
      New Guinea, 374, 656
      New Hebrides, 32
      Pacific area, 205, 496, 996
  blood in, 206
  and Colorado tick-fever, diagnosis, differential, 118
  complications, neurologic, following, 996
  diagnosis, differential, from
    Colorado tick-fever, 118
    tsutsugamushi disease, 992
  immunity, production of, 891
  -like fever, in U.S.A., 804
  symptoms, 205
  taste, altered, in, 374
  transmission
    Aëdes scutellaris hebrideus, 32
    aircraft, control of, 641
  treatment by thiamin, 997
  and yellow fever, relationship between, 804
Dental survey of Marshall Islands, (420)
Depopulation
  in Belgian Congo, 846
    Nkundo tribe, 75
    Tshuapa district, 76
Dermacentor sp., control by spraying, 151
Dermatitis
    in Wales, among troops, 940
  caused by
    Cymbopogon citratus, 66
    Ochrogaster contraria, treatment, 66
    rodent mites, 940
DERMATOLOGY AND FUNGOUS DISEASES, 66–67, 142–143, 225–226, 310–313, 485–486, 584–587, 654–656, 750–751, 831–834, 927–928, 1026–1030

Dermatomycoses, treatment by organic mercurials, 142
Dermatoses
in Malaya, 831
New Guinea, 656
Pacific area, 1027
Solomon Islands, 657
fungous, in Marshall Islands, 420
treatment, 1027
vitamins, 832
Diarrhoea in Assam tea estates, 233
Dichlor diphenyl trichlorethane, *and see under* Insecticides
estimation of, in tissues and body fluids, 153
insecticidal action of, 152
insecticides containing, composition of, 659
persistence of, in oil-bound water-paint, 152
pharmacologic action of, 153
toxicity of, for goldfish and frogs, 256
Dientamoeba fragilis, 386
infection, in U.S.A., in children, 644
Dioctophyma renale infection, in Brazil, 817
Diphtheria
complicating typhus, 266
cutaneous, (935)
Diphyllobothrium sp.
in Eire, 906
Wales, 906
infection, *see under* Tapeworm Infection
parasitizing trout, life cycle of, 397
Diptera
densities of, (231), 760
larvae and pupae, respiration of, 150
Dipylidium caninum, in dogs, in China, 742
Dirofilaria immitis
in dogs, in China, 742
infection, experimental, treatment by radioactive antimony, 747
microfilariae, periodicity of, in dogs, 52
Diseases
of Assam, 154
Burma, 154
India, 154
Madagascar, (233)
Middle East, 154
communicable
in China, 1048
French Colonies, 1038
transmitted by Arthropods, in Texas, (229)
cutaneous, differential diagnosis from pulmonary acariasis, 320
deficiency
in Federated Malay States, 138
Hong Kong, 137
atypical
detection of, 307
treatment, 307
fungous, of skin, treatment, comparative values of, 1028
haemolytic, siderocytes in, 1023
insect vectors of, (662)
respiratory
in Solomon Islands, 657
treatment by sulphadiazine, 657
skin
in Turkey, 310
relation of climate and race to, 310
tropical
of Pacific area, 319
control of, 762
Diseases—*cont.*
Stitt's, Diagnosis, Prevention and Treatment 7th edition [STRONG] (book review), 421
in returned soldiers, 318
in Tropics, Diagnosis and Treatment of [TROWELL] (book review), 1050
Dropsy, in Assam tea estates, 233
Drug addicts
malaria in, 621
pellagra in, (822)
Drugs in tissues, fluorescence microscopy for demonstration of, 415
Dyromys nitedulus, as possible reservoir of relapsing fever, 564
Dyschromia, spirochaetosis associated with, in Argentine, 832
DYSENTERY
amoebic *see* **AMOEBIASIS**
BACILLARY, 34–36, 121–124, 206–207, 284–285, 379–381, 559, 728, 808–809, 893–894, 997–998
in Africa, North, in British prisoners of war, 121
Asia, South East, 753
Assam tea estates, 233
Australia, 835, 893
China, 1048
India, 381, 408
Marshall Islands, 420
Middle East, 5, 147, 380, 728
in American troops, 206
New Guinea, 559, 656, 808
U.S.A., 379
carriers, in Algeria, 34
complications, 894
diagnosis, differential, from appendicitis, 997
fatty diarrhoea in, chronic and relapsing treatment, 123
giardial dysentery simulating, 129
pathogenesis, 380
prophylaxis, bacteriophage, 997
in returned soldiers, 319
splenomegaly, 380
symptoms, 380
treatment
bacteriophage, 997
sulphonamides, 559, 753
comparative value, 124
phthalylsulphathiazole, 35
sulphaguanidine, 122, 284, 319, 656, 728, 753, 808, 893
sulphanilyl benzamide, toxicity and absorption of, 207
sulphapyridine, 408
in troops, 284
complicating kala azar, 101
giardial
in Abyssinia, 1002
Africa, East, 1038
Brazil, (209)
Fiji, in child, 897
India, 468, 897
Morocco, in children, 420
New Zealand, in naval personnel, 468
South Africa, in child, 897
U.S.A., 287
in children, 644
in children, 287, 420, 644, 897

Dysentery—*cont.*
giardial—*cont.*
pseudo-epilepsy due to, (810)
simulating Asiatic cholera and bacillary dysentery, 129
symptoms, recurrent rectal haemorrhages, 897
treatment, 897
acranil, 909
butyl acridine derivative, 468
mepacrine, 129, 288, 897, 1002
stovarsol, 129
Echinochasmus perfoliatus, in dogs, in China, 742
Echinococcosis *see* Hydatid disease
Echinococcus granulosus, in dogs, in China, 742
Embadomonas intestinalis infection
in Morocco, in children, 420
U.S.A., in children, 644
Endolimax nana infection
in East Africa, 1038
Morocco, in children, 420
U.S.A., in children, 644
Entamoeba
coli infection
in East Africa, 1038
U.S.A., in children, 644
histolytica
cultivation of, 999
cysts, 37, 40
destruction of, 286
effect of
chlorine and ozone on, 40
ultra-violet rays on, 999
flies as carrier of, 759
water-borne, destruction of, by synthetic detergents, 734
growth, influence of cholesterol and vitamins on, 36
variability of, 730
invadens as aid in study of *E. histolytica*, 322
Enterica, *see also* Typhoid fever
in U.S.A., 379
Ryle's stomach tube in, 146
Enterobius vermicularis
eggs,
effect of temperature on, 579
under finger nails, 925
in house dust, 925
infection
in Africa, East, 1038
Mexico, in children, (926)
Morocco, in children, 420
New Zealand, in naval personnel, 468
Panama, in children, 578, 911
U.S.A., 400, 920
in children, 644
appendicitis and, 137
in children, 420, 578, 644, (926)
diagnosis, 921, 922, 924
NIH swab, 578
symptoms, 578
treatment
gentian violet, 578, 911, 921, (1021)
methylene blue, 400
quassia, 578
tetrachlorethylene, 578
triphenylmethane derivatives, (1021)
pathogenicity of, 400
Enteromonas hominis infection, in Morocco, in children, 420

ENTOMOLOGY
GENERAL, 72–74, 150–153, 229–231, 322–325, 412–414, 499–506, 594–595, 659–662, 757–760, 839–843, 938–941, 1042–1046
medical
in Venezuela, 322
of military importance, in Canada, (229)
Entomological services and malaria control, (229)
Entomologists and the sanitary corps, (229)
Eosinophilia
in South Pacific, 49
accompanying transitory lung infiltrations, (411)
benign
in India, 754
with pulmonary shadows, 754
in cerebrospinal fluid, in U.S.A., 816
tropical
in British Guiana, 497
India, 408, 411, 589, 590
Samoa, 838
aetiology, (149), 589
cold agglutination in, 411
filariasis and, relationship between, 837
symptoms, 589, 590
treatment, 837
arsenic, agranulytic angina during (838)
Epilepsy
in Belgian Congo, 836
pseudo-, due to giardial dysentery, (810)
susceptibility of coast and mountain dwellers to, (327)
Eretes sticticus, habits of, 323
Eristalis causing intestinal myiasis, (843)
Erythredema, eosinophilic, in Palestine, 590, 591
Espundia *see* **LEISHMANIASIS,** muco-cutaneous
Eutrombicula alfreddugesi, relation of, to disease, 505
Eye diseases
in Egypt, 1031
India, 1036
South Africa, 1036
riboflavin deficiency as cause of, 1036
treatment
penicillin, 487, 488, 489, 1031, 1036
use of iontophoresis to increase concentration of, 489
sulphonamides, 490, 491, 1031
tissue, 491
Fasciola hepatica
infection, in Argentine, (816)
survival *in vitro*, 474
Favism
in U.S.A., 61
blood in, 61
Fever
epidemic, with eosinophilia, 411
pretibial, in U.S.A., 804
Filarial
lymphangitis
in Belgian Congo, 820
Brazil, 647
inguinal glands in, 820
nodule, simulating cyst of external semilunar cartilage, 305
Filariasis, (577), (919) *see also* Wuchereria, Loa, Onchocerca, Dracunculus, Dirofilaria
in Brazil, 647

Filariasis—*cont.*
in British Guiana, 915
China, 747
Ellice Islands, 478
Pacific area, 53, 304, 319, 478, 496, 819
Porto Rico, 941
Samoa, 478, 479, 480
U.S. service personnel, 478, 1020
Virgin Islands, 53
diagnosis, 304
laboratory, 915
and eosinophilia, tropical, relationship between, 837
genital manifestations in, 53
heterophile agglutinins and cold autohaemagglutinins in, 941
histopathology of, 480
lymphatic system, lesions of, 305
prophylaxis, 1020
symptoms, 304
transmission, 647
Aëdes scutellaris, 53
treatment (including experimental), 1021
anthiomaline, 54
toxic reactions, 54
neostam, 136
neostibosan, 136
X-ray, 918
Fish
larvivorous, of Punjab, comparative value of, (92)
poisoning
in Hawaii, 593
Mariana Islands, 592
poisons, 321
Flacurciaceae, characters of, 214
Fleas
of Canada, Alberta, 805
rearing of, 323
Flies
control, 1044
domestic, as carriers of intestinal parasites, in China, 759
of North China, feeding preferences of, (842)
of West China
breeding habits of, (842)
natural enemies of, (842)
Fluids, test for presence of pyrogenic substances in, 415
Gangrene, in typhus, 267
General paralysis of the insane, in Belgian Congo, 836
Geophagia, in animals, (943)
Giardia intestinalis infection *see* Dysentery, giardial
Ginkgo-nut poisoning, in China, in children, 756
Glossina
of Nigeria, 790
Tanganyika, 702, 703
Uganda, 13
control
in Sudan, 969
pyrethrum, 503
hybrids, 98, 99
hypertrophied salivary glands in, 868
Glossina
austeni, infection of, with *T. congolense*, 703
brevipalpis
activity of, 14
variation in, (356)
Glossina—cont.
fuscipleuris, infection of, with *T. congolense* 703
longipennis, infection of, with *T. congolense* 703
morsitans
activity of, 14
analysis of population, in Tanganyika, 186
life economy of, (14)
pallidipes, (187)
activity of, 14
with hypertrophied salivary glands, susceptibility of, to trypanosome infection, 868
salivary gland infection of, 14
palpalis
rearing of, experiments, (791)
palpalis fuscipes
breeding
away from water, 98
places of, 13
Glossitis in China, 651
Gnathostome infection of eye, in India, 919
Goitre, in Belgian Congo, 836
Gonorrhoea, *see also* Venereal Diseases
in Angola, 319
Australia, 835
Belgian Congo, 75
Marshall Islands, 419
Pacific area, 496
treatment by sulphonamide, 496
Granulocytopenia, acute, complicating kala azar 101
Granuloma
coccidioidal *see* coccidioidomycosis
inguinale *see* Ulcerating granuloma of pudendum
nasal, due to *Rhinosporidium seeberi*, (1028)
venereum *see* Ulcerating granuloma of pudendum
Graomys griseoflavus centralis, reservoir of rural plague, 33
Gynaecology, malaria and, (6)
Haemaphysalis sp.
in dogs, in China, 742
leporis-palustris, isolation of filter-passing agent from, 375
HAEMATOLOGY, 58–62, 141, 222–224, 308–309, 403, 482–484, 582–583, 651, 652, 750, 822–827, 1022–1023
Haemoglobinometry, clinical, 1022, 1023
Haemoglobinuria *see under* **BLACKWATER FEVER**
Health
in India, 941, 942
conditions, in Marshall Islands, 419
education, in South African rural native schools 416, 417
hygiene and, of African labourers, 319
Notes for Government Officials [TRESTO (book review), 764
public practice, adaptation of, to foreign cultures, 417
services
national, for Ceylon (1049)
in Portuguese Colonies, reform of, 762
Heart
diseases
in Belgian Congo, 836
Porto Rico, 836
altitude and, 67
-water disease, in South Africa, 980

Heat
acclimatization to, 226
exhaustion, 313, 493
in Iraq, 492
treatment, 144
resistance of African natives, 317
HEAT STROKE AND ALLIED CONDITIONS, 143–146, 226–228, 313–318, 492–494, 587–588, 751–753, 928–932
in Iraq, 492
symptoms, 493
treatment, 144
uraemia and, 588
Heat
syndrome, in Middle East, 147
work in, affected by intake of water, salt and glucose, 226
HELMINTHIASIS, 45–55, 133–137, 215–220, 299–306, 395–402, 473–482, 569–580, 646–650, 741–748, 815–821, 905–926, 1015–1021, *see also* under names of worms
in Africa, East, 1038
Marshall Islands, 420
Morocco, in children, 420
Pacific area, 49
diagnosis, differential, from pulmonary acariasis, 320
eosinophilia and, 49
Summary of Recent Abstracts, 610–614, 667–674
Helminths
carrier rate of, in foodhandlers, in South African hospital, 894
control, in U.S.S.R., River Moskva, 569
rôle of, in verminous appendicitis, 473
Hemiplegia, in chronic malaria, 87
Hepatitis, infective
in Asia, South East, 754
Middle East, 147
amoebiasis and, 319, 382
blood, erythrocyte sedimentation rate in, 250
diagnosis, differential, from tsutsugamushi disease, 992
Hippelates spp., as possible vectors of pinta, 1027
Hippoboscus spp., in dogs, in China, 742
Histoplasmosis
in U.S.A., 654
diagnosis, 655
spontaneous, in dog, 486
symptoms, 654
treatment, 655
Hookworms
Ancylostoma duodenale
eggs, flies as carriers of, 759
larvae, in treatment of polycythaemia, 913
penetration of skin of hamster by, 135
Necator americanus, polysaccharide fraction isolated from, 45
Housing
animal, in tropics, (78)
tropical
in Queensland, 76
physiological principles and, 76
Hydatid disease
in Argentine, 910
New Zealand, (742)
Hydatid disease—*cont.*
of bone, 911
in Chile, 573
U.S.A., 817
treatment, 817
of heart, (477)
of kidney, 911
of liver, 910
of lung, (476), 910
symptoms, 475
treatment, 476
of peritoneum, 911
of spleen, 911
Hydatids associated with choleperitoneum, 399
Hygiene, Institute of, Morocco, report 1943, (233)
Hymenolepis infection *see under* Tapeworm infection
Hypoderma bovis infection in man, 842
Impetigo, bullosa
prophylaxis, 1027
in tropics, 1026
Inermicapsifer cubensis, 477
Infant mortality, in India, 941
Infants, breast-fed, supplementing of, 140
Insect physiology and toxicology, enzyme activity as factor in, (941)
Insecticides, 660, 1044, 1045
anabasine sulphate, 1045
benzyl benzoate, 799
constitution and toxicity of, 499
contact, mode of entry, 869
containing D.D.T., composition of, 659
D.D.T., 152, (153), 182, 270, 499, 500, 537, 661, 757, 758, 785, 839, 841, 842, 880, 1044, (1045)
double-nozzled spray for application of, 1044
mode of entry on contact, 869
dimethyl phthalate, 502
Euphorbia dendroides, 502
Indalone, 502
MYL, 270
Neocid, 500, 501
pyrethrum
aerosol, 759
mode of entry on contact, 869
synergists, 660
Rutgers, 612, 502
Insects
control
at army camps, (229)
sprays, 499
feeding of, on laboratory animals, apparatus for, (74)
and health of military personnel, (229)
physiology and toxicology of, enzyme activity as factor in, (941)
repellents for, 660
vectors of disease, (662)
Insanity from *Catha edulis*, 658
Intestinal parasites, flies as carriers of, in China 759
Intussusception complicating amoebiasis, (286)
Iodomoeba bütschlii
infection
in Africa, East, 1038
Morocco, in children, 420
U.S.A., in children, 644
ingestion processes, 938

Isospora hominis infection
in Mexico, 594
New Zealand, in naval personnel, 468
Ixodes sp.
control by spraying, 151
persulcatus
bionomics of, 324
hibernation of, 324
Japanese river fever, *see* **TYPHUS GROUP OF FEVERS,** mite-borne, tsutsugamushi disease
Jaundice
following mapharsen therapy, with sprue-like syndrome, 402
infective, in Pacific area, 496
spirochaetal, cardiac involvement in, 210
Jungle sores, *see under* **ULCERS, TROPICAL**
Juxta articular nodes
in Jamaica, 131
treatment, 131
Kala azar, *see under* **LEISHMANIASIS**
Keratitis
herpetic
malaria and, 1035
treatment by sulphadiazine, 1035
interstitial, treatment by penicillin, 1036
Keratoconjunctivitis, in India, 1037
LABORATORY PROCEDURES, 232, 414–416, 506, 662, 843–845, 941, 1046–1048
Larvicides, *see under* Anopheles, Mosquitoes, control
Latrodectus
geometricus, 405
indistinctus
in South Africa, 404
bionomics, 404
var. *karrooensis*, 405
Leishmania
cultivation, 543
braziliensis
action of sulphonamides on, 20
cultures, effect of *p*H changes on, 361
chagasi as synonym of *L. donovani*, 359
infantum, inoculation of, into chorio-allantoic membrane of chick embryo, 872
tropica
cultivation, in chick embryo, 454
cultures, infectivity of saline suspensions of, 360
LEISHMANIASIS, 17–20, 100–102, 190–191, 262–264, 356–361, 454–455, 542–544, 635, 711–713, 875–877, 976–979
in Brazil, 712
Panama, 544
Venezuela, 191
canine, in Eritrea, 977
cutaneous
in Middle East, 19
Panama, 359
localization of, 454
treatment
antimon-ditoxin, 712
berberine sulphate solution, 20
ethyl chloride, 20
neostam, 20
sequelae, 20
X-ray, 20
Leishmaniasis—*cont.*
diagnosis, slide agglutination, 977
of frambesial type, 544
kala azar, (876), (977)
in Assam, 17, 100
tea estates, 233
Brazil, 876
China, (100), 101, 875
Colombia, 976
Eritrea, 100
India, 262, 263, 357, 358, 409, 542, 543, 557, 711, (777)
Malta, 876
Sudan, 18, 190, 875
U.S.S.R., 356
U.S. troops, 635
Venezuela, 323
in children, 263
cold haemagglutinins, 454
cold-susceptible globulin fraction in, 262
complications
dysentery, 101
granulocytopenia, 101
necrosis of jaws, 101
noma, 101
pneumonia, 101
in *Cricetus auratus*, treatment, comparative value of, 19
diagnosis, 711
complement-fixation test, 358
sternal puncture, 101
in dogs, skin changes in, (876)
epidemiology, (102)
experimental, 262
infantile
in Algeria, 101
Spain, 978
treatment
antimonial derivative, (102)
pentastib, 101
toxic reactions following, 101
sodium antimony gluconate, 978
malaria and, 543
and Relapsing Fever [GUASCH] (book review), 426
transmission, (102)
in India, 357
congenital in *Trichosurus vulpecula*, 358
treatment (including experimental), (102), 635
neostam, 101
neostibosan, 101
penicillin, 978
phenamidine, (264)
sodium antimony gluconate, 17
V, 263
sodium salt of mannite antimonic acid, (876)
stilbamidine, 101
toxicity of, 18
solustibosan, (876)
urea stibamine, 101
typhus and, 557
of lymph glands, (876)
lymph nodes in, 976
muco-cutaneous
in Sudan, 190
U.S.A., 712
Venezuela, 191, 323
diagnosis, Montenegro skin test, 979

Leishmaniasis—*cont.*
muco-cutaneous—*cont.*
in monkey experimentally infected with Sudan kala azar, 875
mutilating forms of, 264
treatment by antimon-ditoxin, 712
oriental sore, 359, *see also* **LEISHMANIASIS,** cutaneous
in Africa, North, 455
India, 543, 978
Palestine, 361
Sudan, 190
experimental, 877
trauma and, 876
treatment
mepacrine, 543
sulphonamide, 455
umbellatine, use of, 191
vaccination, 360, 361
length of interval before immunity, 102
Summary of Recent Abstracts, 429–433
treatment
aromatic amidines, colour reaction for, 264
penicillin, 544
visceral, transmission, experimental, breeding of sandflies and, (876)
LEPROSY, 43–45, 132–133, 212–214, 290–299, 389–395, 471–473, 566–569, 646, 736–741, 813–814, 899–904, 1004–1018 *see also Mycobacterium leprae*
in Antigua, 390
Argentine, 902, 1012
Australia, 835
Belgian Congo, 75, 394, 741
in pygmies, 295
Bolivia, 813
Brazil, 296, 472, 567, 901, 1006
British Guiana, 737
China, (814)
Cuba, 44, 45, 212, 568
Dutch West Indies, 391
Europe, (814)
Fiji, 294, 736, (814)
Hawaii, 296, (814)
India, 290, 291, 393, 738, 739, 1005, 1014
Jamaica, 390
Malaya, (814)
Marshall Islands, 420
Nevis, 390
Nigeria, 392, 1013
Northern Rhodesia, 392
Pacific area, 496
Porto Rico, 941
Philippine Islands, (814)
St. Kitts, 390
Senegal, (214)
South Africa, 292
Switzerland, 389
U.S.A., 646, 737, (814)
acid-fast
bacteria isolated in, 566
and alcohol-fast organism obtained from human lesion, 1006
culture of, 1006
alopecia in, (1010)
British Empire Leprosy Relief Association, Indian Council of, annual report 1944, 1005
programme of, 1004

Leprosy—*cont.*
census, in Cuba, (44)
in children of leprous parents, 212, 1006
classification of, 43
Claude-Bernard-Horner syndrome in, 567
control, 290, 291
in Belgian Congo, V.A.I.L. system, 741
British Guiana, Mahaica Hospital, 737
British Colonies, 1004
Fiji, Makogai Leper Hospital, 294, 736
India, 1014
Nigeria, Oji River Settlement, 392
Owerri Province, 1013
Northern Rhodesia, Luapula Settlement, 392
South Africa
Emjanyana Leper Institution, 294
Mkambati Leper Institution, 292
Pretoria Leper Institution, 292
U.S.A., National Leprosarium, 737
cultivation of acid-alcohol-fast bacilli from blood-sucking insects fed on lepers, 132
diagnosis, 296
Mazzini flocculation slide, Kolmer-Wassermann and Kahn standard tests, compared, 297
skin reactions, 1010
euglobulin in, 297
experimental, from gland-juice of leper, 1006
heterophile agglutinins and cold autohaemagglutinins in, 941
immunological reactions in examination of child contacts in, 471
infection of Tachinidae, 1008
lepra reaction during thyroid medication for obesity, (394)
leprolins
and lepromin, comparative skin tests, 133
preparation of, 132
Souza-Araujo
and lepromin, comparison between, 1010, 1011
preparation of, 1010
lepromin
and leprolins, comparative skin tests with, 133
test
in non-endemic countries, 900
prognostic value of, 568
sensitization to, 297
lesions, pathology of perforating ulcer of sole, 473
Mitsuda test *see* lepromin test
nasal mucosa in, histopathology of, 567
neural
in Fiji, 295
infectivity of, 295
neuritis, trigeminal, simulating, 901
ocular
lachrymal gland in, 901
lesions, treatment, 1013
onychalgia and, 568
in rats, 473
in Belgian Congo, 741
Brazil, (904)
effects of prolonged administration of sapotoxin on, 814
effect of vitamin B_1 deficiency on, 814
geographical distribution of, 903

Leprosy—*cont.*
in rats—*cont.*
lepra cell in, 394
skin lipoids in, (214)
transmission, 395, 903, 1014
treatment
sulphanilamide, 298
sulphapyridine, 903
Research Department, School of Tropical Medicine, Calcutta, report 1943, 739
serum reactions in, significance of, 739
skin, pathological reaction of, 295
as social and economic problem, 298
Summary of Recent Abstracts, 851–856
sympathectomy in, 740
symptoms, early, 1009
synonymy in, (214)
syphilis and, 296
tracheotomy in, 901
transmission
(attempted) to splenectomized monkeys, 900
cockroaches as possible means of, 294
conjugal, 294
experiments, 1009
ticks and insects, experiments, 1007, 1008
treatment, 45, 392, 472, 737
chemotherapy, 1012
diphtheria toxoid, 44
experiments, (1013)
Grenz rays, 740
hydnocarpus oil, 737
iodized, preparation of, 393
and sodium gynocardate, 741
oil of citronella, 394
penicillin, 646
plasma transfusion, 297
sulphanilamide, 739
sulphapyridine, failure of, 1005
sulphathiazole, 902
tuberculoid, 738, 1010
in Cuba, 44
treatment by diphtheria toxoid, 44
ulcers, treatment by sulphanilamide, 298
xanthelasma and, 214
Leptospirosis
in France, Normandy, 288
French West Africa, 289
Hawaii, 42
U.S.A., 130, 565
immunity, 277
muscle lesions in, 565
symptoms, 131
treatment (including experimental), 131
penicillin, 210, 211, 288, 469
sodium, 288
streptomycin, 898
whole blood transfusions, 43
Leukaemia, eosinophilic
in India, 410
diagnosis, differential, from pulmonary acariasis, 320
Lice
behaviour of, 151
control, (595), 660
D.D.T., 537
pyrethrum synergists, 660
laboratory rearing of, 594
Limnatis nilotica infection, 396
Litomosoides carinii, microfilarial periodicity of, 820
Liver *and see under* **AMOEBIASIS**
abscess complicating amoebiasis, 319
aspiration biopsy apparatus and technique 1046, 1047
cirrhosis, with ascites, in Jamaica, in children 755
dehydrated, preparation of, 826
Lizards, poisonous, of the world, 62
Loeffler's syndrome
in Belgian Congo, 836
South Africa, 755
U.S.A., in negress, 149
ascariasis and, 1039, 1040
creeping eruption and, 1041
symptoms, 1039, 1040, 1041
treatment by emetine, 755
trichiniasis and, 148
Lymphogranuloma inguinale *see also* Venereal diseases
in Africa, 211
Marshall Islands, 419
Macasus rhesus, blood picture of, (141)
Madura foot, 751
actinomycosis of urinary bladder complicating 312
Maduramycotic lesions, experimental, 833
MALARIA, 3–10, 85–95, 170–185, 244–257, 335–353, 433–451, 533–541, 614–633, 684–700, 778–787, 856–868, 959–968
in Africa, 410
Central, 690
East, 964
French Equatorial, (172)
North, 5
Spanish West, 180
West, 6, 438, 690
Algeria, (171), 445
Angola, 319
Arabia, 335
Asia, South East, 753
Assam
in children, 434
tea estates, 233
Australia, 85, 835
Belgian Congo, 172
British Guiana, 614, 615, 785
Bulgarian army, 92
Burmese refugees, 962
Ceylon, (245), (620), 684, 959
Far East, 696
Finland, 621
Great Britain, (434)
Greece, 891
Grenada, 86
India, 178, 338, 408, 442, 444, 618, 6[illegible] 620, 625
in child, 87
Indochina, 335
Iran, 248, 533
Kenya, 959
Mexico, 177
Middle East, 5, 147
Morocco, (6)
New Guinea, 244, 656, 690
Nigeria, 786
Pacific area, 319, 496, 536, 630, 6[illegible] 698, 962, 963
Panama, 176, 338, 690
Porto Rico, 941

Malaria—*cont.*
in Sierra Leone, 434
Solomon Islands, 341, 658, 690, 962
Spain, 171
Sudan, 246
Switzerland, 684
Trinidad, 617
Turkey, in children, 860
U.S.S.R., 254, 628
in children, 339, 349
U.S.A., 173, 620, 626
U.S. troops, 629
Venezuela, 322, 697
age frequency in, 170
anaemia
reticulocytes in, 338
treatment, 338
antigens, normal human stromata as, for complement fixation in sera, 689
appendicitis and, (964)
atypical, in Morocco, (6)
avian
in chicks, effect of riboflavin deficiency on, 540
in ducks, 540
mixed infection, 867
effect of dietary changes on, 864
exoerythrocytic stages in, effect of sulphonamides on, 699
relapse mechanism in, 92
treatment
quinine and mepacrine, inhibitory effect of pyridoxine on activity of, 699
sodium iodobismuthate, 866
substituted quinoline and acridine compounds, 451
sulphonamides, 353, 787
tyrothricin, 449
blackwater fever and, 409, 451, 701
blood
erythrocyte sedimentation rate in, 250
sensitivity of, to haemolysis, 700
on board an LST, 857
carriers, treatment by quino-plasmoquine, 92
cerebral
in India, 691, 861
in U.S. troops, 340
Middle East, 147
diagnosis, 861
Ryle's stomach tube in, 146
symptoms, 340, 341, 691
treatment, 340, 341, 691, 753
methyl-sulphonate-mepacrine, 862
quinine, 7, 861
in children, 87, 339, 349, 434, 860
symptoms, atypical, 339
treatment, 349
complications, typhoid fever, 962
control, 185, (864) *and see under* Anopheles, prophylaxis
in Algeria, 698
America, North and South, 87
British Guiana, 785
Bulgarian army, 92
Gold Coast, 255
Greece, 91
Nigeria, 786
entomological services and, (229)

Malaria—*cont.*
control—*cont.*
on impounded waters, 183, 184
policy for rural Africa, 965, 966
Practical. A Handbook for Field Workers [GUNTHER] (book review), 849
U.S. Public Health Service programme, (229)
diagnosis, 438
buffer precipitation test, 861
intradermal reaction, 174
mistaken, 962
radiographic splenic index, 5
Treatment and Prophylaxis [BISPHAM] (book review), 945
in drug addicts, 621
dynamics of, malariometric values, (622)
effect of, on serologic tests for syphilis, 440
epidemics, forecasting of, in India, 620
eradication of, by destruction of sporogonic cycle of Plasmodium, 686
experimental, contributions of, to knowledge of human malaria, 185
forecasts, in Ceylon, relation of plasmodia findings in blood films to, 959
gynaecology and, (6)
hemiplegia in, 87
heterophile agglutinins and cold autohaemagglutinins in, 941
at high altitudes, 959. 961
immunity, (533)
Intermediary Hosts of, in Netherlands East Indies [TAYLOR] (book review), 511
Investigations Service of Medical Department of British Guiana, Report for 1943, 614
kala azar and, 543
and Relapsing Fever [GUASCH] (book review), 426
kodachrome photomicrography in. 441
liver involvement in, 341
in lizards, 541
in monkeys, treatment by
stilbamidine, 537
thiazole derivatives of sulphanilamide, 448
nephritis and, 176, (439)
nephrosis in, (439)
neuritis and, 963
noma and, 860
Pan-American Commission, 2nd Report of, 87
pathological lesions of brain, 250
pernicious
in U.S.S.R., 628
pathology, 628
symptoms, 628
treatment
mepacrine, 628
quinine, 628
Plasmodium ovale, persistence of, 434
pneumonitis and, 176
prophylaxis, (171), 694
atepe, 180
chemo-, 179
Diagnosis and Treatment [BISPHAM] (book review), 945
mepacrine, 7, 254, 349, 350, 631, 632, 657, 785
indirect determination of plasma, 779
morphological changes in parasite during, 85

Malaria—*cont.*
prophylaxis—*cont.*
observations on, 695
urinary excretion of, factors affecting, 780
versus quinine, 88
pamaquin, 254
plasmoquine, 632
quinine, 180, 349, 350, 619, 631, 632, 657
sulphate, hypoprothrombinaemic action of, 964
sulphonamides, 631
versus mepacrine, 88
psychogenic, in Pacific area, 691
relapses, 753
Plasmodium vivax strain, clinical features of, 692
treatment, 176, 341
mapharside, 697
mepacrine, 692
quinine pamaquin, 693
sulphadiazine, 698
resembling cholecystitis, (862)
in returned soldiers, 318
Ryle's stomach tube in, 146
serum in, *Bact. typhosum* Vi agglutinins, content of, in, 250
sex frequency, 170
spleen
spontaneous rupture of, 438, (533), (628), (964)
traumatic rupture of, (250)
splenic enlargement, radiological estimation of, 4
infarcts, 689
study of, in Venezuela Department of Public Health, 868
Summary of Recent Abstracts, 155–165, 235–243
symptoms, 5
neuropsychiatric, 963
ocular, 439
syphilis and, blood and spinal fluid tests for, 690
terminology in, 244
therapeutic
in U.S.A., 174, 175
parasite density in, 175
Plasmodium vivax, periodicity of, 686
renewed activity in, 174
treatment, by penicillin, 446
transmission, 433
blood transfusion, 174
bank, 627
control of, 688
experimental, *A. culicifacies*, 624
treatment, 6, 171, 176, 694
arsenic, 91
atabrine *see* mepacrine
atebrin *see* mepacrine
berberine, use of, in, 178
burasaine, 964
butyl acridine, 444
Calgluquina, 177
Chinese drugs, comparative value, 630
cinchona bark alkaloids, plasma concentration following, 178
Diagnosis and Prophylaxis [Bispham] (book review), 945
drug control, 533

Malaria—*cont.*
treatment—*cont.*
estimation of serum atebrin concentration 443
mapharside, 697
mepacrine, 6, 92, (253), 318, 339, 409, 438 442, 695, 696, 701, 753, (863)
adsorption-fluorescence, estimation of, 78
derivatives of, in urine, 8
determination of
in blood, 8
direct, in plasma and blood, 782
effects of, 628
on isolated respiratory enzymes, 252
on liver, 783
on oxygen consumption of tissues, 252
on thiamin deficiency, 784
eosinophilia caused by, 630
estimation of, in urine, 781, 863
excreted, optical activity of, (349)
excretion of, 251
hippuric acid liver test in relation to, 536
indirect determination of plasma, 780
mechanism of elimination of, 348
mental symptoms following, 9, 253
methyl-sulphonate intramuscularly, 862
pharmacological basis for, 343
pharmacology of, 443
pigmentation of sclera, 252
prolonged, effects of, 862, 863
tolerance of, nutrition and, 8
toxic psychoses following, 629
toxicity of, 783
versus quinine, 88
mepacrine and mapharsen, failure of, 696
mepacrine and quinine, 6
mepacrine and sulphathiazole, 443
pamaquin, toxic effects of, 253
pamaquin naphthoate, 701
plasmoquine, (253), 434
blackwater fever following, 409
causing haemolytic anaemia, (351)
haemoglobinuria following, (351)
toxic symptoms following, 434
premaline S, 445
quinacrine, 178, 445
cerebral symptoms following, (784)
quinine, 87, 92, 338, 339, 438, 442, 701, (863
absorption experiments, 534
blindness following, 91
causing haemolytic anaemia, (353)
effect on isolated respiratory enzymes, 25
intramuscularly, 862
intravenously
antagonists for circulatory depressio of, 964
influence of, on myocardium, 442
mechanism of elimination of, 348
soft tissue calcification from, 779
sulphate, hypoprothrombinaemia, actio of, 964
versus mepacrine, 88
quinine and mepacrine, 6
quinine and chhatim, 177
quinine and sulphathiazole, 443
quinoline No. 31, gamostatic efficiency o 351
solvochin, 536
stilbamidine, adsorption-fluorescence, esti mation of, 783

Malaria—*cont.*
treatment—*cont.*
sulphathiazole
and atebrin, 443
and quinine, 443
totaquine, 442
equivalent quinine index and efficiency of, 250
production of, 251
variations in, 5
in wartime, 433
zooprophylaxis in, (967)
Malnutrition *see also* **DEFICIENCY DISEASES**
in Assam tea estates, 233
Bantus, 306
India, 407
Manson, Sir Patrick
centenary of, 510
work of, 845
Mastomys coucha, structure of prostate glands in, 281
Medical organization in Belgian Congo, 419
Medicine
in jungle warfare, 656
native, in Southwest Pacific, (327)
Recent Advances in, Symposium on (book review), 765
tropical
in Middle East 1940–1942, (595)
Manual of [MACKIE *et al.*] (book review), 665
Meningitis
in Australia, 835
cerebrospinal
in Angola, 320
Assam tea estates, 233
Ryle's stomach tube in, 146
treatment by sulphapyridine, 233
Meningo-encephalo-myelitis in white mouse, due to *T. gambiense*, 258
Mepacrine, photofluorometric determination of, 90
Metagonimus yokogawai in dogs, in China, 742
Meteorology of Belgian Congo, (849)
Microfilaria streptocera in Belgian Congo, 821
Microfilariae, isolation of, from blood for antigen, 916
Milk, human, intoxication due to B_1 avitaminosis, 139
MISCELLANEOUS, 760–763
MISCELLANEOUS DISEASES, 67–71, 146–150, 318–322, 406–412, 495–498, 589–594, 656–659, 753–757, 835–838, 935–938, 1038–1042
Mites
of New Guinea, 991, 993
occurrence in human sputum, 73
rodent, causing dermatitis, 940
Moniliformis moniliformis, in Toulon rats, 137
Mosquitoes
of Australasian Region, Keys to, including Synopsis of their Distribution and Breeding Habits [KNIGHT *et al.*] (book review), 511
Ellice Islands, 478
Fiji, 938
New Jersey and their Control [HEADLEE] (book review), 963
Samoa, 479
Venezuela, 641
Anopheline of Australasian Region (book review), 1049
Mosquitoes—*cont.*
attacks, activity in, 230
collecting, programme of 7th Service Command 1942–1943, (504)
control, 939
anti-larval siphons, (446)
D.D.T., 182, 537, 757, 785
-thanite sprays, 632
drainage, 246
swamp, 786
dusting, pyrethrum, 447
engineering works, 619
fish breeding, 246, 619
larvicides
D.D.T., 182, 537, 757, 785
toxicity of, for goldfish and frogs, 256
oil-emulsion, 73
mineral, 632
Paris green, 246, 351, 619
phenothiazine, 32
oiling, 246, 619
spraying, 180, 255, 619
thiamin chloride as repellent, 73
trapping, 72, (940)
disease-carrying, recording of sounds produced by, 661
larvae, mounting of, (413)
and leprosy transmission, 1009
Mumps
in African native, 70
Australia, 835
with cerebral symptoms, 70
with orchitis, in Pacific area, 496
scrub typhus simulating, in Australia, 28
Musca
domestica vicina,
pupation of, 413
vicina, life history of, (842)
Mycobacterium
leprae
cultivation, 212, 900
pleomorphism in, 213
decolorizing of, 295
non-acid fast-forms of, 899
leprae murium, experimental infection of *Amblyomma cajennense* with, (904)
Mycosis, cutaneous
in Algeria, 67
caused by *Phialophora verrucosa*, 67
Myiasis
in Uganda, 230
conjunctival
in Uruguay, (230)
caused by *Oestrus ovis*, (230)
creeping, human, 842
furuncular, 151
intestinal, 413
due to Eristalis, (843)
lesionary, 230
caused by *Chrysomyia inclinata*, 230
pyodermic, in Costa Rica, in children, 843
Myocarditis, bilharzial, Stokes-Adams syndrome due to, 396
Myositis, tropical
in India, 935
treatment by sulphathiazole, 935
Naja sore
in India, 405
treatment, 405

Necator americanus
infection *see under* Ankylostomiasis
penetration of skin of hamster by, 135
Necrosis of jaws complicating kala azar, 101
Nematodes causing intestinal perforation, (219)
Nephritis
malaria and, 176, 439
war, trench fever and, 204
Nephrosis in malaria, 439
Neuritis
malaria and, 963
trigeminal simulating leprosy, 901
Nicotinamide methochloride, elimination tests, 926
Nicotinic acid restriction in man, 821
Noma
in Gold Coast, 290
complicating kala azar, 101
malaria and, 860
treatment by penicillin, 290
Nursing, Tropical, Aids to [COCKER] (book review), 79
Nutrition, relationship of, to mepacrine tolerance, 8
Ochrogaster contraria, dermatitis caused by, 66
Oedema of leg and foot, in East African native, 71
Oestrus ovis causing conjunctival myiasis, (230)
Onchocerca volvulus microfilaria, silver staining of, 819
Onchocerciasis
in Guatemala, 748
Mexico, 649, 748
U.S.A., 480
diagnosis, biopsy, 54
and *Wuchereria bancrofti* infection, diagnosis, differential, 481
Onyalai
in South Africa, 148
haemorrhagic bulla in, 148
Onychalgia, leprosy and, 568
Ophthalmia
gonorrhoeal, treatment by penicillin, 1036
neonatorum, treatment by penicillin, 488
snake venom, 583
Ophthalmology, penicillin in, 1036
OPHTHALMOLOGY, TROPICAL, 486–492, 1030–1037
Oriental sore *see under* **LEISHMANIASIS**
Ornithodorus
erraticus, Tunisian strain of, refractory to *S. hispanica*, (811)
moubata
in Belgian Congo, 810
abnormal development of, (151)
turicata, transmitting relapsing fever, in California, 387
Oropsylla spp., distribution of, in North America, 805
Ostertagia ostertagi
characteristics of, 50
infection, in U.S.S.R., in man, 50
Oxyuranus scutellatus bites, fatal, (225)
Oxyuriasis *see* Enterobius infection
Pancreatitis caused by Ascaris, 304
Papillitis, malarial, in New Guinea, 1035
Pa ping
in China, 69, 70
symptoms, 69, 70
treatment, 69, 70
Paracoccidiosis
in Venezuela, 655
symptoms, 655
Paralysis
Kiating, 69, 70
tick, pathogenesis of, (324), 414
Paraplegia following rabies vaccination, 169
Parasites
of dogs, in China, 741
intestinal
in China, (41)
in children, techniques for determination of, 644
metazoan, immunity to, 326
of sheep, 912
Paragonimus sp. in dogs, in China, 742
Pasteur Institutes
abroad during the war, (326)
Algeria, report 1943, (75)
French West Africa, report for 1942, (326)
Pasteurella pestis
in aerated media, 727
ribonuclease activity of, 281
Pedicinus albidus, infection of, from typhus carrying monkeys, 547
Pediculus humanus
in Venezuela, 323
capitis, in Venezuela, 323
Pellagra, 307
in China, (927)
Egypt, 57
Hong Kong, 138
U.S.A., 56, 57, 482, (822)
blood and urinary nicotinic acid in, (927)
diagnosis, 57
in drug addict, (822)
electrocardiographic changes in, 821
gastroprival, experimental, causation of (308)
infantile, in South Africa, 748
nicotinic acid deficiency in, (749)
oesophageal manifestations in, 57
in patient receiving vitamin B complex, 56
treatment, 57
methionine, 749
niacin, 821
ventriculin, 749
Pemphigus
allergy as cause of, 311
foliaceus, in Brazil, 311
Penicillin, Indications for its Use and Method of Administration (spec article), 525–528
Perlèche, 308
Phialophora
jeanselmei, isolated from mycetoma of hand, 833
verrucosa, causing mycosis, 67
Phlebotomus spp.
in India, Hyderabad Province, 979
Portugal, (1044)
U.S.A., California, (151)
Venezuela, 191, 323
biology, (1044)
control
D.D.T., 758
dimethyl phthalate, 119
" K-preparation ", 940
incidence, in China, Szechwan, (100)
n.sp., in Algeria, (662)

Phlebotomus papatasii, reaction of, to light, 230
Physiology of the African, effect of sun's rays on, 508
Piedra of hair tips
in Argentine, 586
caused by *Trichosporon ribeiroi*, 586
Pinta
in Brazil, 928
Cuba, 750
Mexico, 143
initial lesion in, 584
Kahn triple quantitative verification technique in serology of, 927
Spirochaeta herrejoni in case of, 928
transmission
experimental, 585
Hippelates spp., 1027
treatment by penicillin, 143
PLAGUE, 32–34, 280–282, 375–377, 466, 558–559, 642–643, 724–727, 805–806, 892 *see also Pasteurella pestis*
in China, 724, 726, 1048
India, 558
Madagascar, 280
Pacific area, 319
Peru, (466)
U.S.A., 32
chronic, in grey rats, 558
control, 726
in U.S.A., 282
experimental, 466
treatment by penicillin, 642
immunization of animals to, 377
pneumonic
in Madagascar, 280
U.S.A., 33, 805
decline of, 726
recovery, 805
treatment by sulphadiazine, 805
prophylaxis, E.V. vaccine, 280
in rats
in Venezuela, 281
and cats, in Argentine, 33
and human, relationship between, 725
Summary of Recent Abstracts, 528–533
sylvatic
in Argentine, (34), 466
transmission
Graomys griseoflavus centralis, 33
Thrassis spp., (281)
vectors of, 805
transmission, 375, 376
X. cheopis, mechanism of, 33
Mastomys coucha, 281
treatment
serum, 725
sulphadiazine, 558, 725
sulphathiazole, 725, 892
vaccination, opsonic index as test of immunity, 642
vaccine
E.V., 806
living, experimental studies, 726
reactions to, 377
Planorbis spp. in Belgian Congo, (815)
Plants
Emergency Food and Poisonous, of Islands of the Pacific (book review), 234
poisoning from, in South Africa, 153
Plasmodia
biochemistry of, 967
exoerythrocytic cycle of, (968)
growth and multiplication of, *in vitro*, 867
inactivation of, by X-rays, 623
morphological changes under mepacrine prophylaxis, 85
nomenclature of, 244
sporogenic cycle, malaria eradication by destruction of, 686
staining, Wright's, 232
Plasmodium
boissyi
growth, 1017
life span, 1017
self-fertilization in, 1018
brasilianum, asexual reproduction of, effect of immunity on, 256
cathemerium, effects of high temperatures on, 94
durae, exoerythrocytic stages of, 10
elongatum, nuclei, structure of, 185
falciparum, developing gametocytes and schizonts of, 338
gallinaceum
development of, 538
experiments with, 865
in vitro metabolism of, 448
new mosquito hosts for, 633
serial passage of, in chick embryos, 865
sporozoites, inoculation of chick embryos with, 539
knowlesi antigens, 352
lophurae, mosquito transmission of, 352
ovale, 756
in New Guinea, 244
persistence of, 434
relictum of pigeon, mosquito transfer of, 95
tenue, in Ceylon, (245)
vivax
" band " forms of, in Ceylon, (245)
Chesson strain, 628
Feulgen reaction and, 857
strains, peculiarities of, 622
study of, under electron microscope, 85
Pneumoconiosis, differential diagnosis from pulmonary acariasis, 320
Pneumonia
in Angola, 319
Australia, 835
Pacific area, 496
complicating kala azar, 101
Ryle's stomach tube in, 146
Pneumonitis, malaria and, 176
Poisoning
from *Catha edulis*, 658
datura
in Eritrea, 498
Tanganyika, 498
Uganda, 497
fish, 321
in Hawaii, 593
Mariana Islands, 592
ginkgo-nut, in China, in children, 756
plant, in South Africa, 153
stramonium, 756
Polyarthritis, anaemia, sickle-cell and, 141
Polycythaemia, treatment, administration of *A.duodenale* larvae, 913
Polyneuritis following jungle sores, 934

Population, problems of,
heavy, 507
of India, 506
Protozoa
flagellate
cytology, 838
morphogenesis, 838
Human, Manual of, with special reference to their Detection and Identification (book review), 327
intestinal
in East Africa, in troops, 1038
Mexico, in children, 36, 594
Morocco, in children, 420
carrier rate of, in foodhandlers, in South African hospital, 894
diagnosis for, 594, 809
flies as carriers of, 759
staining, tannic acid fixative method for, 937
PROTOZOOLOGY, GENERAL AND PROTOZOAL DISEASES, 71–72, 228–229, 322, 412, 594, 838–839, 937–938
Medical, Knowles's Introduction to, 2nd edition [Das Gupta] (book review), 427
Psychiatric syndromes, treatment by nicotinic acid, 581
Psychiatry among West African troops, 508
Psychosis, Ryle's stomach tube in, 146
Public health services of Morocco, (233)
Pyodermia
in Turkey, 310
in tropics, (1027)
Pyrexia in wartime, diagnosis and management of, 409
Pyrogens, detection of, in fluids, by biological methods, 121
Quinine
blindness, 91
derivative (QDP), structure of, 9
distribution of, in fluids and tissues of dogs, 778
estimation of, in blood, 90
and quinidine content of blood, estimation of, 697
and stilbamidine, effect of, on reticuloendothelial system, 943
RABIES, 165–170, 674–683
in Canada, in child, 676
French West Africa, 678
Kenya, 678
U.S.S.R., 167
autolysis, 674
control, 167
in Great Britain, 680
diagnosis, histologic, 676
immunity, natural, 677
international conference on, need for further, 679
pseudorabies of Aujeszky, 682, 683
reintroduction into Great Britain, danger of, 680
research work on, at Rockefeller Institute, 170
treatment, 167
vaccination, 167
complications following, 168
paraplegia following, 169
vaccine, production of, 678
Rabies—*cont.*
virus
cultivation, in embryo of laboratory animals experiments, 166
street and fixed, comparison of, 169
Radiology, effect of climate on equipment, 848
Rat-Bite fever, (130), (1003)
in Northern Ireland, 42
U.S.A., 811
due to *Streptobacillus moniliformis*, 42
experimental, 645
following bite of kitten, 1002
symptoms, 1002
treatment
neoarsphenamine, 1003
penicillin, 811, 1003
RELAPSING FEVER AND OTHER SPIROCHAETOSES, 41–43, 130–131, 209–211, 288–289, 387, 469–470, 564–566, 644–645, 734–736, 810–812, 898–899, 1002–1003, *see also* Spirochaeta
in Abyssinia, 469
Angola, 319
China, 735, 1048
Eritrea, (42), 102
Libyan Desert, 42
Mozambique, 209
Panama, 564
South Africa, 41
U.S.S.R., 130, 565, 644, 898
Venezuela, 323
complications, neurological, 42
experimental
immunity, 734
treatment, penicillin
intracranially, effect of, on brain involvement, 735
relationship to brain involvement, 387
latent, with rheumatoid manifestations, 130
Malaria and Kala Azar [Guasch] (book review), 426
spirochaetes, 564
carriage of, by tick vectors, 644
symptoms, meningitic, 735
transmission, 130
Ornithodorus
rudis, 323
turicata, 387
treatment (including experimental), 209, 469
neoarsphenamine, 564
streptomycin, 898
virus, *Dyromys nitedulus*, as possible reservoir of, 564
Renal syndrome, cause of, 968
REPORTS, SURVEYS AND MISCELLANEOUS PAPERS, 75–78, 153–154, 232–234, 326–327, 416–421, 506–510, 595–596, 845–849, 941–944, 1048–1049
Reptiles, poisonous, of the world, 62
Reticulo-endothelial system, effect of quinine and stilbamidine on, 943
Rheumatism in Australia, 835
Rhinoscleroma, in Brazil, 935
Rhinosporidiosis, 1042
in Cuba, 225
Rhinosporidium seeberi, causing nasal granuloma (1028)

Rhipicephalus spp., 505
sanguineus, naturally infected with Rocky Mountain fever, 721
Rhodomyrtus macrocarpa causing blindness, 68
Riboflavin
deficiency *see* ariboflavinosis
nutrition, in man, 749
urinary excretion of, 650
Rickettsiae *see under* **TYPHUS GROUP OF FEVERS**
Rickettsial infections in animals, 980
Rocky Mountain fever *see under* **TYPHUS GROUP OF FEVERS,** tick-borne
Russian headache fever *see under* **TYPHUS GROUP OF FEVERS**
SANDFLY FEVER, 117–119, 641–642
in Middle East, 147
Palestine, (996)
control, dimethyl phthalate as repellent, 119
immunity, 277
suspected, in Porto Rico, 642
Sarcophaga fuscicauda, life history of, (842)
Scabies, in Marshall Islands, 420
Schistosoma mansoni
defective granular eggshell formation by, 133
polysaccharide fraction isolated from, 45
Tropicorbis havanensis as possible intermediate host of, 646
Schistosomiasis
in Africa
East, 1038
French West, (215)
Egypt, 1017
Madagascar, (215)
Porto Rico, 941
Southern Rhodesia, 815
Togoland, (215)
associated with cancer of liver, 592
control, in Egypt, 1017
heterophile agglutinins and cold autohaemagglutinins in, 941
in returned soldiers, 319
splenomegaly (Egyptian) and, 906
spread, *Bulinus forskalii* as factor in, 815
treatment by antimony, 815
urinary
in Morocco, 395
South Africa, 46
diagnosis by cystoscopy, 46
symptoms, 46, 395
treatment, 46, 395
vectors of, in Belgian Congo, (815)
water treatment and, (896)
Schistosomiasis haematobium
in Angola, 1015
India, 742
treatment by stilbamidine, 572
Schistosomiasis japonicum, (906)
in Leyte Island, 1018
U.S. naval personnel, 1019
symptoms, 1018
treatment
chemotherapy, 1019
fouadin, 1019
tartar emetic, 1019
urticaria and, 1018
Schistosomiasis mansoni
in Brazil, 572, 1016, 1018
Kenya, 215
Schistosomiasis mansoni—*cont.*
in Porto Rico, 572
in troops, 47
U.S.A., in Arabs, 47
Venezuela, 47
asymptomatic, 47
blood picture in, 572
Banti's syndrome due to, 646, 815
blood in, 1016
in children, 215
creeping eruption in, 215
electrocardiogram in, 572
histology, 1016
pulmonary
in Argentine, 46
complications, 47
diagnosis, 47
treatment by antimonials, 47
treatment
antimony, 1016
fouadin, 47
Schistosomophora hydrobiopsis, vector of schistosomiasis, in Leyte Island, 1018
School medical service, Morocco, report 1943, (234)
Scorpion sting, treatment, 141
Screwworm infestation of nasal passages, (1044)
Scurvy
in Eritrea, 140
treatment, 140
Sea water, effects of drinking, 316
Simuliidae
of New World, (230)
[VARGAS] (book review), 663
Venezuela, 323
effect of light on adults, 919
larvae, factors affecting the fixation of, 919
population, considerations on, 919
Siphunculata, behaviour of, 151
Skin
epigastric, differential rôles of layers of, on diffusion rate of water, 752
loss of water from, measurement of, 752
Sleeping sickness *see* **TRYPANOSOMIASIS,** human, African
Smallpox, in China, 1048
Snails of Belgian Congo, (1017)
Snakes
bite, treatment, 63, 64
poisonous
of Africa, West, 64
Pacific area, 63
Peru, 64
the world, 62
Vipera aspis, toxicity of serum of, 1026
SPECIAL ARTICLES, 517–525, 525–528, 597–609, 947–959
Spider bites, *Latrodectus mactans*, symptoms, 65
Spirocerca sanguinolenta in dogs, in China, 742
Spirochaeta
duttoni, inoculation of, into chorio-allantoic membrane of chick embryo, 872
euygyrata infection, in Morocco, in children, 420
gallinarum, cultivation, 735
herrejoni, finding of, in case of pinta, 928
hispanica, transmitted by intra-nasal instillation, 130
persica, transmission, hereditary, of, in *O. erraticus*, failure of, 811

Spirochaetosis, dyschromia associated with, in Argentine, 832
Spironema persica, annular forms of, 469
Splenomegaly, Bengal, 936
Sporotrichial lymphangitis, treatment, 586
Sporotrichosis, in Cuba, 586
SPRUE, 220–222, 402, 749, 1021–1022
in Australia, 749
Brazil, 222
India, 409
U.S.A., 1021
diagnosis, 222, 749
-like condition
in Asia, South East, 754
India, 220, 221
intrahepatic jaundice with, 402
non-tropical, (222)
serum potassium in, 222
syndrome of unusual aetiology, (1022)
vitamin levels, 1021
Stagnicola bulimoides techella, bionomics of, (302), (474)
Staining
solutions, self-buffering, 232
Wright's, for malaria parasites, in thick blood films, 232
Stains, Romanovsky, deterioration of, in various organic solvents, 662
Stilbamidine and quinine, effect of, on reticulo-endothelial system, 943
Stings
jelly-fish, 937
sting-ray
in India, 937
toxicity of, 937
Stokes-Adams syndrome, due to bilharzial myocarditis, 396
Stramonium poisoning, 756
Streptobacillus moniliformis, causing rat-bite fever, 42
Strongyloides spp., (1020)
stercoralis infection
in Africa, East, 1038
European, (304)
Hungary, 49
Panama, in children, 911, 912
Solomon Islands, 658
South Pacific, 49
U.S.A., 135
complicated by tuberculosis, 135
eosinophilia and, 49
of genito-urinary tract, 576
pulmonary infiltration in, 592
symptoms, 49
treatment, 50
gentian violet, 135, 912
hexylresorcinol, 911
Strongylosis, renal, in Brazil, 817
Sun's rays
effect on man, 317, 508
physiology of the African, and, 508
Suva, Central Medical School, annual report 1943, 416
Sweat glands, congenital absence of, 145
Syphilis *see also* Venereal Diseases
in Belgian Congo, 75, 836
Pacific area, 496
diagnosis, serologic tests, effect of malaria on, 440
leprosy and, 296
Syphilis—*cont.*
malaria and, blood and spinal fluid tests fo 690
Syringes, sterilization and care of, 843, 844
Tachinidae infected with leprosy bacilli, 1008
Taenia
eggs, on perianal skin, 134
solium larva, polysaccharide fraction isolat from, 45
Taeniasis *see under* Tapeworm infection
Taeniorhynchus n.sp., (150)
metallicus, rearing of, 412
Tapeworm infection
Diphyllobothrium
dendriticum infection, in trout, 302, 396, 3
latum infection, 658
in Ireland, 218
anaemia, 215
Gastric Secretion of Persons harbouri [HELANDER] (book review), 5
mansoni, in dogs, in China, 742
Hymenolepis
diminuta infection, 658
nana infection
in Morocco, in children, 420
New Zealand, in naval personn 468
U.S.S.R., 573, 575
treatment
comparative values, 574, 575
male fern, 573
taeniasis
in Argentine, 909
China, in dogs, 742
East Africa, 1038
Ireland, 218
Mexico, 134, 398
Morocco, in children, 420
New Zealand, in naval personnel, 468
South Africa, 894
U.S.A., 907
in children, 644
treatment by acranil, 909
Tapeworms, vitamin requirement of, 302
Thalassaemia *see* Anaemia, Cooley's
Thrassis brennani, n.sp., (281)
Thrombosis, cavernous sinus, treatment penicillin, 489
Ticks
of U.S.S.R., 275
bite pyrexia, 231
control by spraying, 151
North American, distribution and hosts 760
Tinea spp.
of Belgian Congo, Majumbe, (834)
treatment by organic mercurials, 142
Tissue sectioning, rapid technique for, 506
Toxocara canis
in dogs, in China, 742
histological reaction of experimental anim when parenterally introduced, 7
Toxoplasma
avian, 839
mammalian, 839
Toxoplasmosis
in U.S.A., 938
infantile
in U.S.A., 71
post-natal infection, 71

Trachoma
in Africa
East, 1032
Italian East, 490
Argentine, in children, 488
Australia, 835
Cyprus, 487
Middle East, 1037
Uganda, 1033
aetiology, 1034
control, 1032
transmission, congenital, 488
treatment, 491
penicillin, 487
sulphonamides, 486, 490, 491, 1030, 1037
surgical, 1033
Trematodes, digenetic, germ cell cycle in, 905
Trench fever *see under* **TYPHUS GROUP OF FEVERS**
Triatoma
infestans, feeding of, (189), (842)
rubrovaria infection of, by *T. cruzi*, 874
Triatominae
of America, North and Central, 542
West Indies, 542
Tricercomonas intestinalis infection, in East Africa, 1038
Trichinella
antibodies, in cerebrospinal fluid, demonstration of, 481
skin tests, comparative results of, in tuberculous and non-tuberculous patients, 926
toxin, in brain, demonstration of, 481
Trichinella spiralis, polysaccharide fraction isolated from, 45
Trichiniasis, 305, (306)
in Germany, 481, 482
Great Britain, (55)
Lebanon, 400
Mexico, 55, 219
South Africa, 401
circulatory lesions in, 482
diagnosis, Bachman intracutaneous test, 55
and Loeffler's syndrome, 148
of muscle, produced in dog, 579
skin tests in general hospitals and tuberculosis sanatoria, 306
Trichodectus canis, in dogs, in China, 742
Trichomonas
foetus
cultures, bacteria-free, use of penicillin for obtaining, 412
inoculation of, into chorio-allantoic membrane of chick embryo, 872
hominis infection
in East Africa, 1038
South Africa, 209
U.S.A., in children, 644
intestinalis infection, in Morocco, in children, 420
vaginalis
behaviour of, in semi-solid medium, 228
cultures, bacteria-free, use of penicillin for obtaining, 412
infection, (229)
in South Africa, (594)
U.S.A., in males, 72
Trichosporon ribeiroi n.sp., 587
causing piedra of hair tips, 586
Trichostrongylus
eggs, 817
infection in U.S.A., 817
Trichostrongylus instabilis infection, in U.S.S.R., in man, 50
Trichuris
eggs, flies as carriers of, 759
trichiura infection
in Morocco, in children, 420
New Zealand, in naval personnel, 468
Panama, in children, 911
South Africa, 894
U.S.A., in children, 644
treatment
gentian violet, 577
hexylresorcinol, 577, 911
Leche de Higueron, failure of, 912
Trombicula spp.
repellents for, experiments, 73
taxonomy of, (231)
Trombicula fletcheri n.sp., 28
Trombiculid, mites
distribution in Queensland and New Guinea, 369
habits of, 369
Trombiculinae
of Asia, 324
Austro-Malayan and Oriental Regions, 28
Tropical
diseases
in Pacific area, 319
in returned soldiers, 318
medicine
in Middle East, 1940–1942, (595)
Manual of [MACKIE *et al.*] (book review), 665
Nursing, Aids to [COCKER] (book review), 79
Ophthalmology *see* **OPHTHALMOLOGY, TROPICAL**
ulcers *see* **ULCERS, TROPICAL**
Tropicorbis havanensis as possible intermediate host of *S. mansoni*, 646
Tropics
acclimatization, metabolic, to, 930
daily physiological measurements in, 227
How to live in [HUNT] (book review), 766
physiology in, 928
urinary colic in, 931
Trypanosoma
brucei
development of, in salivary glands of *G. palpalis*, 96
inoculation of, into chorio-allantoic membrane of chick embryo, 872
T. gambiense and *T. rhodesiense*, diagnosis, differential, 96
cruzi
developmental cycle of, 972
infecting *Triatoma rubrovaria*, 874
inoculation of, into chorio-allantoic membrane of chick embryo, 872
life cycle of, 872
persistence of, in dead insect vectors, 710
equiperdum
arsenic-resistance, development of, 356
transmission of, to duck, 261
evansi, inoculation of, into chorio-allantoic membrane of chick embryo, 872

Trypanosoma—cont.
gambiense
causing meningo-encephalo-myelitis in white mouse, 258
development of, in salivary glands of *G. palpalis*, 96
inoculation of, into chorio-allantoic membrane of chick embryo, 872
and *T. rhodesiense* and *T. brucei*, diagnosis, differential, 96
lewisi, resistance of rats to, effect of X-ray on, (100)
rhodesiense
in Uganda, 13
development of, in salivary glands of *G. palpalis*, 96
T. gambiense and *T. brucei*, diagnosis, differential, 96
vivax, American, Causative Agent of Bovine Trypanosomiasis in Venezuela; comparison with that in Africa [KUBES] (book review), 422
Trypanosomes, drug resistance of, 971
TRYPANOSOMIASIS, 13–17, 96–100, 186–189, 258–261, 354–356, 452–454, 541–542, 702–711, 788–792, 868–875, 969–976
animal
bovine, in Uganda, 791
treatment by phenanthridinium compound, 259, 260
(*T. cruzi*), lesion of sciatic nerve in, 975
T. evansi infection of horse, (189)
avian, 261, 872
in bats, 873
chicks hatched after inoculation into chorio-allantoic membrane, 871
experimental, 189, 872
treatment
phenanthridinium compounds, 258, 259, 260, 704, 709
phenyl arsenoxides, 15
human, African (sleeping sickness)
in Africa,
French East, 710
Spanish West, 15
Angola, 319
Belgian Congo, 14, 75
Gold Coast, 354, 355, 541
Nigeria, 187, 788, 790
Sudan, 969
Tanganyika, 452
Uganda, 13
antibodies, complement-fixing, persisting after cure, 869
blood picture, 15
cerebrospinal fluid in, 15
control, in Nigeria, 788, 790
diagnosis, 453, 970
difficulties of, 188
gland puncture, 188
laboratory, 15
stabilization of antigens used in, 791
sternal puncture, 14
prophylaxis
Germanin, 14
suramin, 453
treatment (including experimental), 453
Anticoman, 355
antimonials
Trypanosomiasis—*cont.*
prophylaxis—*cont.*
toxicity of, 869
effect of cysteine on, 869
trypanocidal activity of, 869
effect of cysteine on, 869
antrypol, 13
and tryparsamide, 789
Bayer 205, 710
Germanin and tryparsamide, 970
orsanine, 710
penicillin, 870
sodium, 453
pentamidine, 354
stilbamidine, absorption of, by trypanosomes, 970
surfen, 355
tryparsamide, 188, 319, 541
nitritoid reaction following, (710)
V-(p-arsenophenyl)-butyric acid, 354
human, American (Chagas's disease)
in Argentine, 17, 792, 874
Bolivia, (875)
Brazil, (875), 973
U.S.A., 710
Venezuela, 17, 711
conjunctiva in, histopathology of, 792
diagnosis
agglutination and complement fixation tests, comparison between, 975
complement fixation with *T. cruzi* cultures as antigen, 974
spleen-puncture, 17
xeno-, 17
histology, 974
history of, 17
sudden death in, 711
symptoms, cardiac, 874
transmission, 972, 973
effect of D.D.T. on vectors, 973
vectors of, in U.S.A., 710
Summary of Recent Abstracts, 329–335
Tsetse Flies, *and see* Glossina
of French West Africa [GLASCHEN] (book review), 512
Tsutsugamushi disease *see under* **TYPHUS GROUP OF FEVERS,** mite-borne
Tuberculosis
in Argentine, 731
Australia, 835
India, 409
Marshall Islands, 419
Pacific area, 496
amoebiasis and, 731
complicating *S. stercoralis* infection, 135
diagnosis, differential from pulmonary acariasis, 320
pseudo-, of lungs, with eosinophilia in India, 410
Typhoid fever
in China, 1048
India, 409
Middle East, 147
complicated by malaria, 862
haemoglobinuria in, (659)
TYPHUS GROUP OF FEVERS, 20–31, 102–114, 191–204, 264–275, 362–373, 455–464, 544–557, 635–640, 713–721, 792–800, 877–888, 979–996

Typhus Group of Fevers—*cont.*
in Africa, North, 22
Brazil, 988
Ceylon, 886
China, 981
Colombia, 265
Germany, 104, 461
Guatemala, 265
India, 407
Italy, Naples, 22
Mexico, (881)
Middle East, 266
Morocco, 199, 267
U.S.S.R., 456, 458, 796, 989
U.S.A., (366)
antibody formation after intravenous injections of killed typhus-proteus bacilli, 108
antigen
non-specific complement-fixing, in embryonic egg tissues, 635
presence of, in normal guineapig, 364
Bullis fever
experimental, 799
Rickettsia, specificity of, 639
serological studies, 203
vectors of, 799
cardiac disturbances in, 104
central nervous system, pathological anatomy of, 638
circulatory disturbances in, 104
complications, 104
diphtheria, 266
gangrene, treatment, 267
surgical, 106
control, 979
convalescence
" after-fever " in, 105
circulatory disturbances in, 105
diagnosis
agglutination test, 197
by detection of specific antigen in blood, 196
Minkeivich's modification of Weil Felix reaction, 459
skin test, 198, 199
slide agglutination test, 882
strengthening of serum agglutinins by ripening *in vitro*, 545
value of investigation of lice for Rickettsia in, 795
Weil-Felix reaction, 981
electrocardiography in, 106
endemic typhus
in China, 719, 1048
Eritrea, 102
Hawaii, 271
India, 29, 30
Mexico, 272
Porto Rico, 990
Salvador, 796
South Africa, 271, 362, 877
diagnosis
complement fixation test, 983
comparison of water-bath and icebox fixation in, 990
in rodents, 551, 552
serological, 637, 877
and epidemic typhus,
antigenic differences, 716

Typhus Group of Fevers—*cont.*
endemic typhus—*cont.*
diagnosis—*cont.*
diagnosis, differential, 362
and Rocky Mountain fever, immunological relationship between, 636
simultaneous infection, in laboratory animals, 990
Rickettsia, isolation of, from rats, rat-fleas and body lice, 719
transmission, *Ctenocephalides felis*, 462
treatment by mepacrine, 990
virus, 547
epidemic typhus
in Brazil, 638, 986
China, 795, 1048
Colombia, 551
Ecuador, 103
Europe, 879
Great Britain, in prisoners of war, 879, 880
Guatemala, 718
India, 881
Marshall Islands, 420
Mexico, 202
Middle East, 364
Persia and Iraq Force, 24
South Africa, 362, 877
Spain, 22, 984, 988
U.S.S.R., 191
in German troops, 26
aetiology, unitary view of, 193
antigens in infected yolk sacs, 457
diagnosis
complement-fixation test, 983, 984
serological, 877
Weil-Felix reaction, 22
and endemic typhus,
antigenic differences between, 716
diagnosis, differential, 362
and Rocky Mountain fever, immunological relationship between, 636
simultaneous infection, in laboratory animals, 990
histopathology of lesions in, 458
of military importance, (102)
morbid anatomy of, 718
pathology, 266
spread, influence of environment on, 22
symptoms, 24, 461, 795
transmission, rôle of droplet infection, 22
treatment, 26, 462
chemotherapy, 638
para-aminobenzoic, 201
penicillin, 880
serum, 364
from convalescent rabbits, 202
vaccination, 268, 270, 880, 881
vaccine
preparation of, 107
tests, 792
virus, response of rat to peritoneal inoculation of, after rabbit-lung passage, 985
exanthematic *see* epidemic typhus
experimental, 269
in body lice, 987
murine and classic types simultaneously, 457
in *Sigmodon hispidus hispidus*, 192

Typhus Group of Fevers—*cont.*
immunity, 714, 715
in inoculated persons, 27
interruptions in course of incidence of, 460
kala azar and, 557
of military importance, (102)
mite-borne
in Pacific area, 319
Solomon Islands, 658
Sudan, 553
blood changes in, 111
control, 111
diagnosis, Weil-Felix reaction, 28
fluid-balance in, 112
in returned soldiers, 319
simulating mumps, in Australia, 28
symptoms, 109, 111
treatment, 110
tsutsugamushi disease, 272, 367, 371, 980
in Asia, South East, 753
Burma, 883, 884
Far East, 887
New Guinea, 369, 371, 555, 656, 882, 991, 993
Pacific area, 274, 720
blood in, 370
chemistry of, 885
complications, 372, 884
cardiac, 556
control, 720
di-butyl phthalate, 656
diagnosis, 798, 883
differential from
dengue, 992
hepatitis, infective, 992
pathology, 798
post-mortem changes in, 554
prevention, 798, 799
symptoms, 370, 371, 555, 797, 798, 882
transmission, experiments, 463
treatment, 274, 370, 372, 555, 798, 995
Trombicula fletcheri as vector of, 993
in troops, 991, 993, 994
vectors and reservoirs of, 367
murine typhus *see* endemic typhus
neuro-pathological syndromes in, 459
ophthalmological findings in, 548
pathological anatomy of, 458
pathology of circulatory system in, (367)
prophylaxis, 715
Proteus OX 19
agglutination, 981
agglutinins to, in serum of pregnant women, 363, 794
specificity of Weil-Felix reaction, 197
relapses, 25
research, 264
Rickettsia
orientalis
isolation of, from XK typhus, in Ceylon, 886
staining of, 879
prowazeki
classification, 193
composition of, 192
cultivation of, in vitelline membrane of chick embryo, 23
in different diluents, 106
pathogenic and antigenic power of, 366
preservation of, in lungs of mice, 193

Typhus Group of Fevers—*cont.*
Rickettsiae
in Ecuador, 103
behaviour of, in various ectoparasites, 713
cultivation, yolk sac, labile toxin in, 23
filterability of, 455
formolized, suspensions of, reactions following intradermal injection of, (795)
growth of, effect of p-aminobenzoic acid, 796
inclusion bodies in association with, 104
multiplication of, effect of enzyme inhibitors and activators on, 362
pulmonary tissue deprival of, 366
staining, 544
structure of, 23
viability of, 455
rickettsial agglutination, 194, 982
Russian headache fever, 496
Ryle's stomach tube in, 146
scrub typhus *see* mite-borne, tsutsugamushi disease
sera, complement fixation in, 718
Summary of Recent Abstracts, 767–778
symptoms, 29, 266
cardiovascular, 199
nervous, 200
tick-borne
in Africa, East, 720
America, 721
South Africa, 362, 877
U.S.S.R., 274, 464
in animals, in South Africa, 980
boutonneuse fever
in Abyssinia, 113
pseudo-, histology of eschar in, 887
diagnosis
complement fixation test, 983
serological, 877
Rocky Mountain fever
in Mexico, 203
U.S.A., 373
epidemic and endemic typhus, immunological relationship between, 636
experimental, treatment by penicillin, 996
R. sanguineus naturally infected with, 721
symptoms, 203
treatment (including experimental), 373, 888
para-aminobenzoic acid, 888
penicillin, 464, 996
spotted fever of Tobia, virus, persistence of, in *O. parkeri*, 799
symptoms, 720
transmission, 715
blood transfusion, 988
experimental research on, (719)
laboratory, 460
treatment, 365
buxation, 989
calcium gluconate, 266
chemotherapy, 22, 878
comparative values, 20
convalescent blood, (106)
luminal, 267
mepacrine, 266

Typhus Group of Fevers—*cont.*
treatment—*cont.*
penicillin, 988
serum, 980
sodium salicylate, 989
strophanthin, 267
sympatol, 267
vaccine, 989
trench fever
in Germany, 204, 462
blood picture, 204
diagnosis, differential, 639
rheumatic nodules in, 113
symptoms, 204
treatment, 31
antimonials, 31
war nephritis and, 204
uraemic condition in, 25
in vaccinated persons, 796
vaccination, 549
Blanc's vaccine, 268
Cox-Craigie vaccine, 980
egg allergy significance in, 890
in Moscow railway workers, 456
experiments, 793
live " strain E " *R. prowazeki* in, 794
neutralizing power of serum following, 107
sera, 461
vaccines, 980
alum-precipitated, 455
bivalent, preparation of, 27
comparative value of, 882
Durand-Krontovskaya mouse-lung, 549
reactions to, 719
use of goat for preparation of, 365
virus
experiments, 109
inactivation of, 986
response of guineapig to peritoneal inoculation with, 104
in wartime, (719)
Weil-Felix reaction, 545, 546
in dogs, in Brazzaville, 995
Tyroglyphus siro in faeces, 325
Ulcerating granuloma of pudenda *see also* Venereal Diseases
in Australia, 835
Ulcers
chronic
in Brazil, 933
treatment, zeno process, 933
corneal, treatment by penicillin, 1036
phagedaenic
in French West Africa, 933
treatment, 933
TROPICAL, 228, 405–406, 494–495, 588–589, 834–835, 932–935, 1037–1038
in Africa, East, 1037
Angola, 320
Gold Coast, 289
India, 405, 932
Iran, 588
Kenya, 406
Nyasaland, 588
Solomon Islands, 657
aetiology, 932
jungle sores
in Burma, 934
diphtheritic infection of, 934
polyneuritis following, 934
Ulcers—*cont.*
Tropical—*cont.*
osteo-periostitis in, (835)
prophylaxis, 932
symptoms, 932
treatment, 406, 588, 834, 932
Almaza Patch in, 1038
penicillin, 289, 495
plaster casts, 228
spinal anaesthesia, 1037
sulphonamides, 320, 494
Umbellatine, pharmacology of, 191
Urticaria
amoebiasis and, 37
schistosomiasis and, 1019
Varicella, in Angola, 320
Variola, in Angola, 320
Venereal Diseases *and see* Gonorrhoea, Syphilis, Lymphogranuloma inguinale and Ulcerating granuloma of pudenda
in Africa, 211
Angola, 319
VENOMS AND ANTIVENENES, 62–65, 141–142, 224–225, 309–310, 403–405, 583–584, 652–654, 827–830, 1026
Venoms
anavenoms, preparation of, 829
bee, 405
catalytic action of copper during oxidation of, (584)
haemolysis, lecithin and, (829)
physiologic action of, (584)
scorpion, 141
Buthus occitanus, 404
effect of, on vasomotor system, 653
Egyptian, 583
Prionurus spp., 404
snake
adder, (404)
Agkistrodon piscivorus, phospholipase of, (584)
Ancistrodon
mokasen, pattern of bite of, 403
piscivorus, pattern of bite of, 403
Bothrops, blood-pressure and cerebrospinal fluid variations following inoculation with, 309
Bungarus fasciatus, haemolysin production from 224
cobra
as analgesic, 583
in treatment of carcinoma pain, (65)
Crotalus
blood-pressure and cerebrospinal fluid variations following inoculation with, 309
atrox, pattern of bite of, 403
Dendraspis spp., 652
angusticeps, anticoagulant properties of, 653
viridis, 309
anticoagulating properties of, 653
Echis carinata, 64
in Eritrea, 827
in antigens of, 827
Micrurus tschudii olssoni, 65
Naja
nigricollis, 583

Venoms—*cont.*
snake—*cont.*
Naja.—contd
tripudians, haemolysin production from, 224
ophthalmia, 583
Trimeresurus barnetti, 65
viper, prothrombin studies, (829)
Vipera
aspis, 1026
berus, 827
spp. of Algeria, 404
spider
Latrodectus, (225)
indistinctus, 404
mactans, 65
Loxosceles, (225)
Vertebrates and Arthropods examined in Investigations of the Epidemiology of Yellow Fever (special article), 597–609
Vibrios, *see under* **CHOLERA**
Vincent's disease, in Peru, 1041
Vitamin
deficiency, in British prisoners of war, 122
requirement of tapeworm, 302
Vitamin A, adult need of, 308
Vitamin B deficiency
associated with skin diseases, treatment by hydrochloric acid, 582
clinical significance of glossitis and cheilosis in, 580
syndrome allied to sprue, 220
Vitamin B_1 deficiency causing human-milk intoxication, 139
Vitamin C
adult need of, 308
concentration of, in sweat, 145
Water
exchange, 227
of rice region, anophelogenic rôle of, 3
Weight loss
insensible, relation of, to water loss from skin and lungs, 752
in man, measurement of, 752
Weil's disease *see* Leptospirosis
Welfare in British Colonies [MAIR] (book review), 944
West Indian Conference, Report 1944, 760
Wuchereria bancrofti
experimental infection of mosquitoes with, 917
infection and onchocerciasis, diagnosis, differential, 481
microfilariae, antigen prepared from, 219
Xanthelasma, leprosy and, 214
Xenopsylla
brasiliensis, bionomics, 504
cheopis
bionomics, 504
control, D.D.T., 758
Xeropthalmia, in Hong Kong, 138
YAWS, 131–132, 211, 289–290, 387–389, 470–471, 645, 736, 812–813, 899, 1003–1004
in Africa, 211
West, 387
Angola, 319
in Australia, 835
Belgian Congo, 75, 736
Brazil, 812, 1003
Yaws—*cont.*
Ellice Islands, 470
Eritrea, 132
Fiji, 470
Gold Coast, 290
Marshall Islands, 419
New Guinea, 899
Pacific area, 496
bone and joint manifestations in, 387
foot, 813
histology, 899
symptoms, 387, 388
bilateral periorbital hyperostosis, 736
treatment, 388
bismuth, 736
mapharsen and bismuth, 645
subsalicylate, 899
penicillin, 290, 388, 470, 812, 1003
YELLOW FEVER, 32, 205, 114–117, 275–280, 373–374, 465, 557–558, 640–641, 721–724, 800–803, 890–891
in Africa, French West, 276, 723, 802
Nigeria, 800
Sudan, in European, 374
control, 116, 117, 558
disinfection of aircraft, 557
Inter-Departmental Committee on, 721
and dengue, relationship between, 804
diagnosis, complement fixation test, 114, 802
Epidemiology of, Vertebrates and Arthropods examined in Investigations of (special article), 597–609
experimental, treatment by choline hydrochloride, 115
immunity, 277
congenital transmission, experiments, 279
jungle
in Colombia, 114
protection tests, 205
in Eritrea, 147
French West Africa, 723
in recently vaccinated, 276
Research Institute, Lagos, annual report 1944, 800
service
in British Guiana, 557
French West Africa, 275
and similar diseases, immunity relations between, 277
Summary of Recent Abstracts, 81–84
in troops, 276
vaccination, 276, 723
egg allergy, significance in, 890
vaccine
aqueous-base, testing of, in Rhesus monkeys, 803
control of, standards for, 890
manufacture of, standards for, 890
preparation of, 275
virus
inoculation of, in kittens, 277
neurotropic, 373
susceptibility of chicks to, 278
prolonged viability of, 465
reactivation of, from attenuated cultures, 205
susceptibility of mice to, 277

INDEX OF COUNTRIES.

EUROPE

Belgium
  Anopheles of, (778)
Bulgaria
  malaria, in army, 92
Cyprus
  blindness, 487
  trachoma, 487
Eire
  Diphyllobothrium spp., 906
    latum infection, 218
  taeniasis, 218
Europe
  leprosy, (814)
  typhus, 879
Finland
  malaria, 621
France
  Culicidae, (594)
  Moniliformis moniliformis, in Toulon rats, 137
Germany
  cysticercosis, 134
  trench fever, 204, 462
  trichiniasis, 481, 482
  typhus, 104, 461
Great Britain
  dermatitis, in troops, in Wales, 940
  Diphyllobothrium spp., in Wales, 906
  trichiniasis, (55)
Greece
  Anopheles control, 447
  blackwater fever, 92
  malaria, 91
Hungary
  Strongyloides stercoralis infection, 49
Italy
  typhus, 22
Malta
  kala azar, 876
Portugal
  Culicidae of, (1043)
  Phlebotomus spp., (1044)
Spain
  Anopheles of, 171, (859)
  Anopheles algeriensis, (859)
  kala azar, infantile, 978
  malaria, 171
  typhus, 22, 984, 988
Sweden
  haemoglobinuria, paroxysmal, paralytic, 60
Switzerland
  Anopheles, 685
  leprosy, 389
  malaria, 684
Turkey
  cheilitis actinica, 310
  malaria, in children, 860
  pyodermia, 310
  skin diseases, 310
U.S.S.R.
  Anopheles of, 254
  blackwater fever, 257
  helminth control, in River Moskva, 569
  Hymenolepis infection, 573, 575
  kala azar, 356
U.S.S.R.—*cont.*
  malaria, 254
    in children, 339, 349
    pernicious, 628
  Ostertagia ostertagi infection, in man, 50
  rabies, 167
  relapsing fever, 130, 565, 644, 898
  ticks of, 275
  Trichostrongylus instabilis infection, in man, 50
  typhus, 191, 274, 456, 458, 464, 796, 989
    in German troops, 26

ASIA

Arabia
  Anopheles, 335
  malaria, 335
Asia
  Trombiculinae, 324
Asia, South-East
  amoebiasis, 753
  anaemia, in Indian troops, 754
  dysentery, bacillary, 753
  hepatitis, infective, 754
  malaria, 753
  sprue-like condition in troops, 754
  tsutsugamushi disease, 753
Assam
  anaemia, on tea estates, 232
  diarrhoea, on tea estates, 232
  diseases of, 154
  dropsy, on tea estates, 232
  dysentery, bacillary, on tea estates, 232
  kala azar, 17, 100
    on tea estates, 232
  malaria
    in children, 434
    on tea estates, 232
  malnutrition, on tea estates, 232
  meningitis, cerebrospinal, on tea estates, 232
Burma
  Anopheles minimus, 180
  diseases of, 154
  jungle sores, 934
  malaria, in refugees, 962
  tsutsugamushi disease, 883, 884
Ceylon
  acariasis, pulmonary, in soldiers, 320
  Anopheles of, 337
  cancer, 149
  health service, national, (1049)
  malaria, (245), (620), 684, 959
  typhus, 886
China
  amoebiasis, 39
  Anopheles minimus, 180
  ariboflavinosis, 650, 651
  ascariasis, 744
  cholera, 1048
  diseases, communicable, 1048
  dysentery, bacillary, 1048
  filariasis, 747
  ginkgo-nut poisoning, in children, 756
  glossitis, 651

China—*contd.*
helminths in dogs, 742
kala azar, (100), 101, 875
leprosy, (814)
pa ping, 69, 70
parasites
of dogs, 742
intestinal, (41)
pellagra, (927)
plague, 724, 726, 1048
relapsing fever, 735, 1048
smallpox, 1048
typhoid fever, 1048
typhus, 719, 795, 981, 1048
Dutch East Indies
Malaria, Intermediary Hosts of [TAYLOR] (book review), 511
Far East
Anopheles of, 623
malaria, 696
tsutsugamushi disease, 887
Federated Malay States
deficiency diseases, 138
Hong Kong
beriberi, 138
deficiency diseases, 137
pellagra, 138
xerophthalmia, 138
India
amoebiasis, 381, 408, 731
of anus and perineum, 730
anaemia, 407
Cooley's, 59
iron-deficiency, in native soldiers, 651
macrocytic, 222
nutritional, 59
in native troops, 822
Anopheles, 180, 618, 619, 623
identification of, 85
blackwater fever, 701
cholera, 119, 120, 282, 643, 892
dengue, 409
diseases of, 154
dysentery
bacillary, 381, 408
giardial, 468, 897
eosinophilia, tropical, 408, 411, 589, 590, 754
eye diseases, 1036
gnathostome infection, of eye, 919
infant mortality, 941
kala azar, 262, 263, 357, 358, 409, 542, 543, 557, 711
keratoconjunctivitis, 1037
leprosy, 290, 291, 738, 739, 1005, 1014
Research Department, School of Tropical Medicine, Calcutta, report 1943, 739
leukaemia, eosinophil, 410
malaria, 87, 178, 338, 408, 442, 444, 618, 619, 620, 625
cerebral, 691, 861
in U.S. troops, 340
malnutrition, 407
myositis, tropical, 935
Naja sore, 405
oriental sore, 543, 978
Phlebotomus spp., of Hyderabad Province, 979
plague, 558
population problem, 506, 507
schistosomiasis, 742
India—*cont.*
sprue, 409
spruce-like disease, 220, 221
sting-ray sting, 937
tuberculosis, 409
typhoid fever, 409
typhus, 29, 30, 407, 881
ulcers, tropical 405, 932
Indo-China
Anopheles minimus, 180
malaria, 335
Iran
Anopheles of, 248, 533
malaria, 248, 533
typhus, in troops, 24
ulcers, tropical, 588
Iraq
heat, effects of, 492
typhus, in troops, 24
Lebanon
trichiniasis, 400
Malaya
dermatoses, 831
leprosy, (814)
Middle East
amoebiasis, 147, 1002
conjunctivitis, 1037
diseases of, 154
dengue, 147
dysentery, bacillary, 5, 147, 728
in U.S. troops, 206
heat syndrome, 147
hepatitis, infective, 147
leishmaniasis, cutaneous, 19
malaria, 5, 147
sandfly fever, 147
trachoma, 1037
typhoid fever, 147
typhus, 266, 364
Mongolia
Anopheles maculipennis messeae, 249
Palestine
amoebiasis, 128, 894
Anopheles of, 247
ascariasis, 745
conjunctivitis vernalis, 1031
erythredema, eosinophilic, 590, 591
sandfly fever, (996)
Persia and Iraq Force
anaemia, macrocytic, in Indian soldiers, 403
Philippine Islands
leprosy, (814)

AFRICA

Abyssinia
boutonneuse fever, 113
Culicine n.sp., (940)
dysentery, giardial, 1002
relapsing fever, 469
Africa
amoebiasis, 410
cancer of liver, in negroes, 150
lymphogranuloma inguinale, 211
malaria, 410
venereal diseases, 211
yaws, 211
Africa, Central
malaria, 690

Africa, East
amoebiasis, 1038
anaemia, sickle-cell, in Bantus, 825
ankylostomiasis, 1038
appendicitis, in natives, 71
ascariasis, 1038
Chilomastix mesnili infection, 1038
Endolimax nana infection 1038
Entamoeba coli infection, 1038
Enterobius vermicularis infection, 1038
Giardia intestinalis infection, 1038
Iodamoeba bütschlii infection, 1038
malaria, 964
mumps, in natives, 70
oedema of leg and foot, in natives, 70
schistosomiasis, 1038
Strongyloides stercoralis infection, 1038
taeniasis, 1038
trachoma, 1032
Tricercomonas intestinalis infection, 1038
Trichomonas hominis infection, 1038
typhus, 720
ulcers, tropical, 1037
Africa, French
diseases, communicable, 1038
Africa, French Equatorial
malaria, (172)
trypanosomiasis, 710
Africa, French West
Anopheles gambiae, 247
leptospirosis, 289
Pasteur Institute, report for 1942, (326)
rabies, 678
schistosomiasis, (215)
Tsetse Flies of [GASCHEN] (book review), 512
ulcers, phagedaenic, 933
yellow fever, 275, 276, 723, 802
Africa, Italian East
trachoma, 490
Africa, North
dysentery, bacillary, in British prisoners-of-war, 121
malaria, 5
oriental sore, 455
typhus, 22
Africa, Spanish West
malaria, 180
trypanosomiasis, 15
Africa, West
Anopheles of, 434
blackwater fever, 10, 12, 451
malaria, 6, 438, 690
psychiatry, 508
snakes, poisonous of, 64
yaws, 387
Algeria
Anopheles of, 698
dysentery, bacillary, carriers, 34
kala azar, infantile, 101
malaria, (171), 445
mycosis, cutaneous, 67
Pasteur Institute, report 1943, (75)
Phlebotomus n.sp., (662)
Viperidae of, 404
Angola
amoebiasis, 319
anaemia, sickle-cell, (1025)
ankylostomiasis, 319
beriberi, 320
gonorrhoea, 319
Angola—*cont.*
malaria, 319
meningitis, cerebrospinal, 320
pneumonia, 319
relapsing fever, 319
schistosomiasis, 1015
trypanosomiasis, 319
tuberculosis, 320
ulcers, tropical, 320
varicella, 320
variola, 320
venereal diseases, 319
yaws, 319
Belgian Congo
adenolymphocele, 820
Agamofilaria streptocerca, 821
Anopheles of, 172, (688)
brochopneumonia, 836
depopulation, 846
in Nkundo tribe, 75
in Tshuapa district, 76
epilepsy, 836
filarial lymphangitis, 820
general paralysis of the insane, 836
goitre, 836
gonorrhoea, 75, 76
heart disease, 836
leprosy, 75, 394, 741
in pygmies, 295
in rats, 741
Loeffler's syndrome, 836
malaria, 172
medical organization, 419
meteorology, (849)
Microfilaria streptocerca, 821
Ornithodorus moubata, 810
schistosomiasis, vectors of, (815)
snails of, (1017)
syphilis, 75, 76, 836
Tinea spp., (834)
trypanosomiasis, 14, 75
yaws, 75, 736
Cameroons
cancer, 936
Egypt
anaemia, Cooley's, in children, 224
eye diseases, 1031
pellagra, 57
schistosomiasis, 1017
Eritrea
ankylostomiasis, 478
datura poisoning, 498
kala azar, 100
leishmaniasis, caninē, 977
relapsing fever, (42), 102
scurvy, 140
snake venoms, 827
typhus, 102
yaws, 132
yellow fever protection tests, 147
Gold Coast
Anopheles of, 255
malaria, control, 255
noma, 290
trypanosomiasis, 354, 355, 541
ulcers, tropical, 289
yaws, 290
Kenya
ankylostomiasis, 48
Anopheles, 959

Kenya—*cont.*
Anopheles, control, 447
malaria, 959
rabies, 678
schistosomiasis, 215
ulcers, tropical, 406
Libya
relapsing fever, in desert, 42
Madagascar
diseases, social and infectious, (233)
plague, 280
schistosomiasis, (215)
Morocco
ascariasis, 420
Balantidium coli infection, 420
Chilomastix mesnili infection, 420
dysentery, giardial, 420
Embadomonas intestinalis infection, 420
Endolimax nana infection, 420
Enterobius infection, 420
Enteromonas hominis infection, 420
Hygiene, Institute of, report 1943, (233)
Hymenolepis infection, 420
Iodamoeba bütschlii infection, 420
malaria, (6)
public health services, (233)
schistosomiasis, 395
school medical service, report 1943, (234)
Spirochaeta euygyrata infection, 420
taeniasis, 420
Trichomonas intestinalis infection, 420
Trichuris infection, 420
typhus, 199, 267
Mozambique
relapsing fever, 209
Nigeria
Acanthocheilonema perstans infection, 649
Glossina of, 790
leprosy, 392, 1013
malaria, 786
trypanosomiasis, 187, 788, 790
yellow fever, 800
Research Institute, Lagos, annual report 1944, 800
Northern Rhodesia
leprosy, 392
Necator americanus infection, 48
Nyasaland
ulcers, tropical, 588
Senegal
leprosy, (214)
Sierra Leone
malaria, 434
Southern Rhodesia
Bilharzia, Control of [MOZLEY] (book review), 516
schistosomiasis, 815
Sudan
Anopheles gambiae, 246
Glossina control, 969
insects of, 1042
kala azar, 18, 190, 875
leishmaniasis, muco-cutaneous, 190
malaria, 246
oriental sore, 190
trypanosomiasis, 969
typhus, 553
yellow fever, 374
Tanganyika Territory
blackwater fever, 185
Tanganyika Territory—*cont.*
datura poisoning, 498
Glossina, 702, 703
population, analysis of, 186
trypanosomiasis, 452
Tsetse Research Department, report, 702
Togoland
schistosomiasis, (215)
Uganda
anaemia, macrocytic nutritional, 827
datura poisoning, 497
Glossina, 13
myiasis, 230
trachoma, 1033
Trypanosoma rhodesiense, 13
trypanosomiasis, 13
bovine, 791
Union of South Africa
amoebiasis, 38, 643, 894
in infants, 285
anaemia, sickle-cell, 652
ankylostomiasis, 894
ascariasis, 894
blackwater fever, (95)
Bulinus forskalii, 815
dysentery, giardial, 897
eye diseases, 1036
health education in rural native schools, 416
heart-water disease, 980
Latrodectus indistinctus, 404
leprosy, 292
Loeffler's syndrome, 755
malnutrition, in Bantus, 306
onyalai, 148
pellagra, infantile, 748
poisoning, from plants, 153
relapsing fever, 41
schistosomiasis, 46
taeniasis, 894
trichiniasis, 401
Trichomonas
hominis infection, 209
vaginalis infection, (594)
Trichuris trichiura infection, 894
typhus, 271, 362, 877

AMERICA, NORTH

America, North
Anopheles of, 87
Argasidae of (book review), 154
cancer of liver, in negroes, 150
Triatominae, 542
typhus, (229)
Canada
entomology, medical, (229)
fleas, of Alberta, 805
rabies, in child, 676
United States
Aëdes aegypti
in Texas, 757
control, 374
amoebiasis, 208, 379, 729
in children, 644
anaemia
Cooley's, 223
macrocytic nutritional, 482

United States—*cont.*
Anopheles, 173
control, 446
of Pacific coast, 437
ariboflavinosis, 482
ascariasis, in children, 644
beriberi, 482
blastomycosis, cutaneous, (67)
Chagas's disease, 710
Chilomastix mesnili infection, in children, 644
coccidioidomycosis, 66, 225, 485, 655
in air personnel, 313
cysticercosis, 816, 907
dengue-like fever, 804
Dientamoeba fragilis infection, in children, 644
diseases transmitted by Arthropods, in Texas, (229)
dysentery
bacillary, 379
giardial, 287
in children, 644
echinococcosis of bone, 817
Embadomonas intestinalis infection, in children, 644
Entamoeba
coli infection, in children, 644
nana infection, in children, 644
enterica, 379
Enterobius vermicularis infection, 400, 920
in children, 644
eosinophilia in cerebrospinal fluid, 816
favism, 61
fever, pretibial, 804
histoplasmosis, 654
Iodamoeba bütschlii infection, in children, 644
leishmaniasis, muco-cutaneous, 712
leprosy, 646, 737, (814)
leptospirosis, 130, 565
Loeffler's syndrome, 149
malaria, 173, 620, 626
therapeutic, 174, 175
onchocerciasis, 480
pellagra, 56, 57, 482, (822)
Phlebotomus in California, (151)
plague, 32, 33
control, 282
pneumonic, 805
rat-bite fever, 811
Rocky Mountain fever, 373
schistosomiasis, in Arabs, 47
sprue, 1021
Strongyloides stercoralis infection, 135
taeniasis, 907
in children, 644
toxoplasmosis, 938
infantile, 71
Trichomonas
hominis infection, in children, 644
vaginalis infection, in males, 72
Trichostrongylus infection, 817
Trichuris trichiura infection, in children, 644
typhus, (366)

AMERICA, CENTRAL

America, Central
Argasidae of (book review), 154
Triatominae, 542
typhus, 721
Costa Rica
myiasis, pyodermic, in children, 843
Guatemala
onchocerciasis, 748
typhus, 265, 718
Honduras
anaemia, sickle-cell, in Carib Indians, 141
Mexico
alopecia, leprotic, (1010)
amoebiasis, 895
in children, 36, 594
cysticercosis, 647
Enterobius infection, in children, (926)
Isospora hominis infection, 594
malaria, 177
onchocerciasis, 649, 748
pinta, 143
protozoa, intestinal, in children, 594
Rocky Mountain fever, 203
taeniasis, 134, 398
trichiniasis, 55, 219
typhus, 202, 272, (881)
Panama
anaemia, sickle-cell, 582
ankylostomiasis, 911, 912
ascariasis, 911, 914
blood pressure, in Cuna Indians, 420
chromoblastomycosis, (587)
Enterobius vermicularis infection, 911
in children, 578
leishmaniasis, 359, 544
malaria, 176, 338, 690
relapsing fever, 564
Strongyloides stercoralis infection, 911, 912
Trichuris trichiura infection, 911
Salvador
typhus, 796

WEST INDIES

Antigua
leprosy, 390
British West Indies
cancer of oesophagus, (592)
Cuba
Argasidae of (book review), 154
dengue, 803
leprosy, 44, 45, 212, 568
census, (44)
pinta, 750
rhinosporidiosis, 225
sporotrichosis, 586
Dutch West Indies
cancer, 592
leprosy, 391
Grenada
malaria, 86
Jamaica
cirrhosis of liver, with ascites, in children, 755
juxta-articular nodes, 131
leprosy, 390
Nevis
leprosy, 390
Porto Rico
filariasis, 941
heart disease, 836
leprosy, 941
malaria, 941
sandfly fever, suspected, 642

Porto Rico—*cont.*
schistosomiasis, 572, 941
in troops, 47
typhus, 990
St. Kitts
leprosy, 390
Trinidad
Anopheles of, 617
pessoai, 437
malaria, 61
Virgin Islands
filariasis, 53
West Indies
Triatominae, 542

AMERICA, SOUTH

America, South
Anopheles of, 87
typhus, 721
Argentine
amoebiasis, 37, 731
anaemia, sickle-cell, (141)
Anopheles, control, (448)
blastomycosis, 143
Chagas's disease, 17, 792, 874
dyschromia associated with spirochaetosis, 832
Fasciola hepatica infection, (816)
hydatid disease, 910
leprosy, 902, 1012
piedra, 586
plague, 892
in rats and cats, 33
sylvatic, (34), 466
schistosomiasis, 46
taeniasis, 909
trachoma, in children, 488
tuberculosis, 731
Bolivia
Chagas's disease, (875)
leprosy, 813
Brazil
Anopheles of
Island of Santo Amaro, (173)
São Paulo, 87
Chagas's disease, (875), 973
dysentery, giardial, (209)
filarial lymphangitis, 45, 647
filariasis, 647
kala azar, 876
leishmaniasis, 712
leprosy, 296, 472, 567, 901, 1006
in rats, (904)
pemphigus foliaceus, 311
pinta, 928
rhinoscleroma, 935
schistosomiasis, 572, 1016, 1018
sprue, 222
strongylosis, renal, 817
typhus, 638, 986, 988
ulcers, chronic, 933
yaws, 812, 1003
British Guiana
Anopheles of, 615, 616, 785
Central Medical Laboratory, Georgetown, annual report 1943, 326
eosinophilia, tropical, 497
filariasis, 915
leprosy, 737
British Guiana—*cont.*
malaria, 614, 615, 785
yellow fever service, 557
Chile
cysticercosis, cerebral, 908
hydatid disease, 573
Colombia
bartonellosis, 889
kala azar, 976
typhus, 265, 551
yellow fever, jungle, 114
Ecuador
typhus, 103
Peru
ariboflavinosis, 927
plague, (466)
snakes, poisonous, 64
Vincent's disease, 1041
Uruguay
myiasis, conjunctival, (230)
Venezuela
Aëdes spp., 323
Anopheles of, 250, 322
carate, 323
Chagas's disease, 17, 711
Culicidae of, 640
of Bolivar, 465
entomology, medical, 322
kala azar, 323
leishmaniasis, 191
muco-cutaneous, 323
malaria, 322, 697
paracoccidiosis, 655
Phlebotomus spp., 191, 323
plague in rats, 281
relapsing fever, 323
schistosomiasis, 47
Simulium spp., 323

AUSTRALASIA

Australasia
Anopheline Mosquitoes of (book review), 1049
Mosquitoes of, Keys to, including Synopsis of their Distribution and Breeding Habits [KNIGHT *et al.* (book review), 511
Australia
amoebiasis, 835
ankylostomiasis, 835
dysentery, bacillary, 835, 893
gonorrhoea, 835
housing, in Queensland, 76
leprosy, 835
malaria, 85, 835
meningitis, 835
mumps, 835
pneumonia, 835
rheumatism, 835
sprue, 749
trachoma, 835
Trombiculid mites of, 369
tuberculosis, 835
typhus, scrub, simulating mumps, 28
ulcerating granuloma of pudenda, 835
yaws, 835
New Guinea
amoebiasis, 808
ankylostomiasis, 743

New Guinea—*cont.*
dengue, 374, 656
dermatoses, 656
dysentery, bacillary, 559, 656, 808
malaria, 244, 656, 690
mites, 991, 993
Trombiculid, 369
Necator americanus infection, 743
papillitis, malarial, 1035
Plasmodium ovale, 244
tsutsugamushi disease, 369, 371, 555, 656, 882, 991, 993
yaws, 899
New Zealand
amoebiasis, 468
ankylostomiasis, 468
ascariasis, 468
dysentery, giardial, 468
Hydatid disease, (742)
Hymenolepis nana infection, 468
Isospora hominis infection, 468
taeniasis, 468
Trichuris trichiura infection, 468

PACIFIC ISLANDS

Ellice Islands
filariasis, 478
mosquitoes of, 478
yaws, 470
Fiji
Central Medical School, Suva, annual report 1943, 416
dysentery, giardial, 897
leprosy, 294, 295, 736, (814)
mosquitoes of, 938
yaws, 470
Hawaii
dengue, 641, 891
fish poisoning, 593
leprosy, 296, (814)
leptospirosis, 42
typhus, 271
Leyte Island
schistosomiasis, 1018
Mariana Islands
fish poisoning, 592
Marshall Islands
amoebiasis, 420
beriberi, 420
cheilitis, 420
dengue, 420
dental survey, 420
dermatoses, 420
dysentery, bacillary, 420
health conditions, 419
Marshall Islands—*cont.*
helminthiasis, 420
leprosy, 420
lymphogranuloma inguinale, 419
scabies, 420
tuberculosis, 419
typhus, 420
yaws, 419
Melanesia
Anopheles, (778)
New Hebrides
dengue, 32
Pacific area
amoebiasis, 319
ankylostomiasis, 49, 319
clonorchiasis, 319
dengue, 496, 996
dermatoses, 1027
dysentery, bacillary, 319
eosinophilia, 49
filiariasis, 53, 304, 319, 478, 496, 819
gonorrhoea, 496
helminthiasis, 49
jaundice, infectious, 496
leprosy, 496
malaria, 319, 496, 536, 630, 692, 698, 962, 963
psychogenic, 691
mumps with orchitis, 496
native medicine in, (327)
plague, 319
pneumonia, 496
schistosomiasis, 319
snakes, poisonous of, 63
Strongyloides stercoralis infection, 49
syphilis, 496
tsutsugamushi disease, 274, 720
tuberculosis, 496
typhus, 319
yaws, 496
Samoa
eosinophilia, tropical, 838
filariasis, 478, 479, 480
mosquitoes of, 479
Solomon Islands
amoebiasis, 657
ankylostomiasis, 658
dermatoses, 657
Diphyllobothrium latum infection, 658
Hymenolepis diminuta infection, 658
malaria, 341, 658, 690, 962
respiratory diseases, 657
Strongyloides stercoralis infection, 658
tapeworm infection, 658
typhus, 658
ulcers, tropical, 657
St. Thomas Island
anaemia, sickle-cell, (1025)

(74132) Wt. P.1914/311 6/46 Hw. G.344

ALPHABETICAL LIST OF AUTHORS OR SOURCES.

d'Abrera, V. St. E., 959.
Alcantara, A. G., 988.
Alisov, P. A. & Kossovaja, E. M., 989.
Anderson, G. A., von Deschwanden, J., Gray, W. A. & Menzies, T. H., 961.
Angevine, D. M., Hamilton, T. R., Wallace, F. G. & Hazard, J. B., 976.
Annecke, A., McCarthy, D. D. & Wilson, D. B., 966.
Arbona, G., 990.
Arbuse, D. I., 968.
d'Arcangelo, D., 1002 *bis*.
d'Avanzo, C. S., 1026.

Balamuth, W. & Sandza, J. G., 999.
Barlow, C. H. & Abdel Azim, M., 1017.
Battelli, C., Coceani, A. & Rossi, M., 977.
Bengtson, I. A., 983.
Berger, E. & Brzezinski, S., 986.
Blake, F. G., Maxcy, K. F., Sadusk, J. F., Jr., Kohls, G. M. & Bell, E. J., 991, 993.
Bland, J. O. W., 1034.
—— & Wilson, R. P., 1031.
Bloss, J. F. E., 969.
Boase, A. J., 1033.
Boquet, P., 1026.
Boshoff, P. H., 1036.
Brazzaville [A.E.F.], 995.
Brown, A., 1022.
—— & Anderson, A. B., 1023.
Bull. U.S. Army Med. Dept., 1019.

Cairo, 1017.
Cardis, F., 1039.
Carmichael, J., 979.
Case, R. A. M., 1023.
Cayer, D., Ruffin, J. M. & Perlzweig, W. A., 1021.
Ceccaldi, J., 995.
Chamberlain, W. P., Jr. & Bronson, L. H., Jr., 1035.
Chaudhuri, R. N. & Rai Chaudhuri, M. N., 962.
Chorine, V., 1013.
Coghill, N. F., 1001.
Cohen, S. S., 981.
Corrêa, R. R. & Ramos, A. S., 961.
Couceiro, A., 975.
Cruz, W. O. & de Mello, R. P., 1020.
da Cunha, A. M., Guimarães, F. N., Leão, A. E. A. & Cardoso, H. T., 1003.
——, Leão, A. E. A., Guimarães, F. N. & Cardoso, H. T., 1003.

Davidson, S., 964.
Damon, S. R. & Johnson, M. B., 990.
Davey, T. F., 1013.
De la Camara, J. P., 984.
Dharmendra & Rajagopalan, S., 1012.
Dick, G. W. A., 1038.
Dreisbach, R. H. & Hanzlik, P. J., 964.

Earle, K. V., 1041.

Fan, J. H., 1048.
Farooq, M. & Qutubuddin, M., 978.
Felix, A., 979.
Fernández Castanys, A., 978.
Fitzpatrick, F. K., 982, 996.
Fox, L. A., 979.
Fuller, A. T., 1048.
Fulton, F., 979.
Fulton, J. D., 978.

Garnham, P. C. C., 959.
Gillman, T. & Gillman, J., 1046.
Giroud, P. & Panthier, R., 985.
Gutmann, M. J., 1031.

Hammond, A. G., 1038.
Hardesty, J. F., 1036.
Harvey, A. M., 963.
Hawking, F., 971.
—— & Green, F. H. K., 1052.
Hodge, I. G., Denhoff, E. & Vander Veer, J. B., 1020.
Hole, N., 979.
Hunt, A. R., 1019.
—— & Bloss, J. F. E., 969.
Hyman, A. S., 962.

Ibarra Perez, R. & Gonzalez Prendes, M. A., 1009.

Jansen, G., 1018.
Jettmar, H. M. & Shigan Li, 981.

Kaplan, A. & Lindgren, A., 996.
King, H., 971.
Kirwan, E. O'G., Sen, K. & Bose, N., 1036.
Kohls, G. M., Armbrust, C. A., Irons, E. N. & Philip, C. B., 993.

Lee, D. J. & Woodhill, A. R., 1049.
Lent, H., 973.
—— & de Oliveira, S. J., 973.
León, A. P., 984.
Leprosy in India, 1005.
Lewis, D. J., 1042.
Lewthwaite, R., 979.
Lewy, R. B., 1035.
Lindquist, A. W., Schroeder, H. O. & Knipling, E. F., 1044.
Linhares, H., 1014.
Linskis, D. Y., 988.
Longley, E. O., 979.
Lopes, C. F. & Laender, J. F., 979.
Lyon, E. & Kleinhaus, E. M., 1040.

Macdonald, G., 965.
Machella, T. E. & Forrester, J. S., 994.
McLaughlin, R. R. M., 1027.
Maegraith, B. G., Havard, R. E. & Parsons, D. S., 968.
Magalhães, A., Coutinho, B., Gouvêa, L., Lucena, D. & Ignacio, L., 1016.
Magalhães, B. F. & Dias, C. B., 1016.
de Magalhães, O. & Rocha, A., 986, 988.
Mariano, J., 1010.
Medical Research Council, 1052.
Miranda. R. N., 1011.
Miterstein, B. & Stern, H. J., 1030.
Mole, R. H., 1023.
Mom, A. M. & Basombrio, G., 1012.
Morton, H. E. & Engley, F. B., Jr., 997.
Muir, E., 1004.
Muniz, J. & de Freitas, G., 975.

Nabokov, V. A., 1045.
Nery Guimarães, F. & Jansen, G., 972.

de Oliveira Castro, G. M., 960.
—— & Mariano, J., 1009.

Parish, H. J., 979.
Payne, A. M. M., 1001.
Pirk, L. A. & Engelberg, R., 964.
Price, E. W., 1037.
Proc. Roy. Soc. Med., 979.

Reed, H., 1032.
Romaña, C., 974.
—— & Dias, E., 974.
—— & Meyer, H., 972.
Rose, T. F., 997.

Sarmento, A., 1015.
Sen, J. B., 1014.
Shama Rao, A., 1005.
Senekjie, H. A. & Lewis, R. A., 977.
Sharp, H. S. & Biggs, R., 1042.
Sherlock, S., 1047.
Silva, R. & Castañeda, M. R. 990.
Silverman, D. N. & Leslie, A., 1001.
Snyder, J. C. & Wheeler, C. M., 987.
Soberón y Parra, G. & León y Blanco, F., 1027.
de Souza-Araujo, H. C., 1006 *bis*, 1007, 1008 *ter*, 1010 *bis*.
—— Mariano, J. & de Oliveira Castro, G. M., 1009.
—— & Miranda, R. N., 1007.
Speck, J. F. & Evans, E. A., Jr., 967.
Spühler, O. & Kartagener, M., 1039.
Starr, D. F., 1044.
Stoll, A. M., Ward, P. A. & Mathieson, D. R., 999.
Stuart-Harris, C. H., 979.
Sulzberger, M. B., Shaw, H. C. & Kanof. A., 1028.
Swyer, G. I. M., 1002.

Theodor, O. H. & Parsons, B. T., 1013.
Thomas, H. M., Jr. & Gage, D. P., 1018.
Tomlinson, W. J., 1025.
Trowell, H. C., 1050.

Ubatuba, F. & Vieira, G., 996.

War Office, 999, 1001.
Winsor, T. & Burch, G. E., 1024.
Wright, D. O. & Gold, E. M., 1041.

Yarygin, N. E. & Nagibina, N. I., 989.

NAPT

TUBERCULOSIS

—a post-war problem for the British Commonwealth.

For information on

- **EDUCATION**
- **RESEARCH**
- **PROPAGANDA**

write to

National Association for the Prevention of Tuberculosis,
Tavistock House North, London, W.C.1.

MEMBERSHIP—TEN SHILLINGS PER ANNUM.

'MOOGROL'

Ethyl Esters of Hydnocarpus Oil
(Stabilised with 4% Creosote)

For the treatment of Leprosy

An effective preparation of hydnocarpus esters, relatively free from local irritant effect and well tolerated by both intramuscular and intradermal routes.

SPECIFY **'MOOGROL'** *with* **CREOSOTE**

BURROUGHS WELLCOME & CO.
(The Wellcome Foundation Ltd.)
LONDON

ISSUED UNDER *the* DIRECTION *of the* HONORARY MANAGING COMMITTEE *of the* BUREAU *of* HYGIENE *and* TROPICAL DISEASES.

BULLETIN OF HYGIENE

Containing abstracts of current medical literature on public health administration, social services, communicable diseases, sanitation, nutrition, industrial hygiene, bacteriology and immunity, and related subjects of public health interest.

ISSUED MONTHLY.

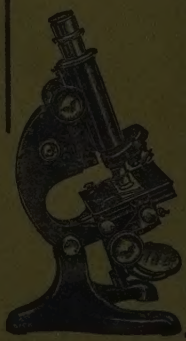
Subscription: 21s. a year, post free.

Orders and Subscriptions should be sent to—

THE BUREAU OF HYGIENE AND TROPICAL DISEASES,
Keppel Street, Gower Street, W.C.1.

BECK MICROSCOPES

For over a century Beck Microscopes have been pre-eminent in design, workmanship and performance. Now available for research, industry, teaching, etc., subject to allocation by Ministry of Supply.



R. & J. BECK Ltd.
69, Mortimer Street
LONDON, W.1